

# ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED BY JAMES PLEASANT PARKER

VOLUME 59

## *Editor*

ARTHUR W. PROETZ, M.D.  
Beaumont Building, St. Louis, 8

## *Associate Editor*

BERNARD J. McMAHON, M.D.  
Missouri Theatre Building, St. Louis, 3

## *Editorial Board*

L. R. BOIES, M.D. . . . .	Minneapolis	MARVIN F. JONES, M.D. . . . .	New York
LOUIS H. CLERF, M.D. . . . .	Philadelphia	HAROLD I. LILLIE, M.D. . . . .	Rochester, Minn.
SAMUEL J. CROWE, M.D. . . . .	Baltimore	JOHN G. McLAURIN, M.D. . . . .	Dallas
W. E. GROVE, M.D. . . . .	Milwaukee	LEROY A. SCHALL, M.D. . . . .	Boston
ANDERSON C. HILDING, M.D. . . . .	Duluth	H. MARSHALL TAYLOR, M.D.,	Jacksonville Fla.
FREDERICK T. HILL, M.D. . . . .	Waterville, Me.	O. E. VAN ALYEA, M.D. . . . .	Chicago

## *Published Quarterly*

BY THE

ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE 1010 BEAUMONT BUILDING, 8

BUSINESS OFFICE P. O. BOX 1345, CENTRAL STATION, 1

ST. LOUIS, MO., U.S.A.

COPYRIGHT, 1950

ANNALS PUBLISHING COMPANY

---

Annual Subscription in United States, Spain, Central and South America, \$10.00 in Advance.  
Canada, \$10.20. Other Countries, \$10.80.

610.5

Ab

09

7/4

# Contents.

	PAGE
I—The Transmission Properties of the Middle Ear. Ernest Glen Wever, Ph.D., and Merle Lawrence, Ph.D., Princeton, N. J.	5
II—Studies on the Histogenesis of the Ampullary Cupula. Thure Vilstrup, Copenhagen, Denmark	19
III—Studies on the Completed Structure and Mechanism of the Cupula. Thure Vilstrup, Copenhagen, Denmark	46
IV—The Physiology of Respiratory Obstruction. John S. Gray, M.D., Chicago, Ill.	72
V—Radium Therapy for Lymphoid Tissue in the Nasopharynx. Edwin B. Bilchick, M.D., and Albert R. Kolar, M.D., New York, N. Y.	78
VI—Radiation Exposure of Personnel Handling the Monel Metal Nasopharyngeal Radium Applicator. Henry J. Rubin, M.D., Barney M. Kully, M.D., and Raymond D. Finkle, Ph.D., Los Angeles, Calif.	90
VII—Evaluation of Irradiation of Pharyngeal and Nasopharyngeal Lymphoid Tissue. Francis L. Lederer, M.D., Chicago, Ill.	102
VIII—Surgical Treatment of Bilateral Abductor Paralysis of the Larynx. John H. Barrett, M.D., Houston, Texas	112
IX—Malignant Melanoma of the Nose and Sinuses. Joseph G. Schoolman, M.D., and Harold W. Anderson, M.D., Chicago, Ill.	124
X—Salivary Gland Tumors of the Submaxillary Gland Associated with Calculi. Ephraim L. Manning, M.D., Davenport, Iowa, and Maurice A. Michael, M.D., Lebanon, Pa.	141
XI—Clinical Electronystagmography. Aram Glorig, M.D., Silver Springs, Md., and Alexander Mauro, New Haven, Conn.	146
XII—Neuralgic Syndromes Associated with Aplasia of the Frontal Sinus. Ettore Giuffrida, M.D., Catania, Italy	152
XIII—Bacteriologic and Clinical Interpretation of the Flora of the Nose and Nasopharynx in Adults. Joseph L. Goldman, M.D., New York, N. Y.	156
XIV—The Carotid Canal, as a Pathway for Extension of Infection in the Temporal Bone. J. G. Druss, M.D., New York, N. Y.	166
XV—Blood Pressure Changes in Fenestration. M. J. Tamari, M.D., and M. H. Cutler, M.D., Chicago, Ill.	179
XVI—Scleroma. Résumé of the Literature. Report of Three Cases. G. W. Olson, M.D., Fresno, Calif.	186
XVII—The Treatment of Bell's Palsy with Histamine. D. A. Skinner, M.D., Newark, Ohio	197
XVIII—Otosclerosis in Identical Twins. A Review and Report of Two Additional Pairs. Arthur L. Juers, M.D., Chicago, Ill.	205
XIX—The Nasal Tip in Rhinoplasty. Use of the Invaginating Technique to Prevent Secondary Drooping. Gustave B. Fred, M.D., Boston, Mass.	215

## CONTENTS—Continued

	PAGE
XX—The Diagnosis and Treatment of Tumors of the Neck. Delmar F. Weaver, M.D., Detroit, Mich.....	224
XXI—Backstage with the Board. Arthur W. Proetz, M.D., St. Louis, Mo.....	231
 <b>Clinical Notes</b>	
XXII—Actinomycosis of the Pharynx and Base of Tongue. Case Report. William Wesley Wilkerson, Jr., M.D., and Lee Farrar Cayce, M.D., Nashville, Tenn. ....	242
XXIII—Fibrosarcoma of the Larynx. Report of a Case in a Child. Daniel Miller, M.D., Boston, Mass.....	246
XXIV—An Unusual Course of an Open Safety Pin in a Baby 8 Months Old. Peter D. Latella, M.D., New Rochelle, N. Y., and David H. Jones, M.D., New York, N. Y.....	254
XXV—Fibroma of the Nasopharynx. A Case Report. Kenneth L. Diehl, M.D., Rochester, N. Y.....	258
 <b>New Instrument</b>	
XXVI—A New Instrument for Performing Tonsillectomy and Other Otorhinolaryngological Operations. Shinji Yoshida, M.D., Fukuoka, Kyushu, Japan .....	262
 <b>Society Proceedings</b>	
Chicago Laryngological and Otological Society, Meeting of Monday, March 7, 1949. Electroencephalography in Diagnosis of Hearing Loss in the Very Young Child—Evaluation of Irradiation of Pharyngeal and Nasopharyngeal Lymphoid Tissue—Blood Pressure Changes in Fenestration Surgery .....	264
Meeting of Monday, April 4, 1949. The Development of the Otic Capsule in the Region of Surgical Fenestration .....	269
Meeting of Monday, November 7, 1949. Favorable Results in Bulbar Polio-myelitis Treated as a Problem in Respiratory Obstruction—Heating of the Human Maxillary Sinus by Microwaves.....	271
Meeting of Monday, December 5, 1949. Physical Laws of the Mechanism Involved in the Removal of Air from the Respiratory Tract under Normal and Abnormal Conditions—Fifty Years of Scientific Progress in Otolaryngology—Outstanding Personalities of the First Half Century of the Chicago Laryngological and Otological Society.....	279
Meeting of Monday, January 9, 1950. Inner Ear Deafness of Sudden Onset—Deafness Following Head Trauma: A Clinical and Experimental Study—Some Unusual Mixed Tumors of the Nose and Throat .....	283
Abstracts of Current Articles .....	294
Books Received .....	301
Notices .....	303
Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation of the American Medical Association.....	306
Officers of the National Otolaryngological Societies.....	307

# ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

---

---

VOL. 59

MARCH, 1950

No. 1

---

---

## I

### THE TRANSMISSION PROPERTIES OF THE MIDDLE EAR

ERNEST GLEN WEVER, PH.D.

AND

MERLE LAWRENCE, PH.D.

PRINCETON, N. J.

The basic function of the middle ear mechanism is well known: it serves as a mechanical transformer, providing for the ready transfer of acoustic energy from the outside air to the fluid of the cochlea. Without such transformer action the vibratory motions of the air particles would not easily be communicated to the heavier particles of the cochlear fluid, but to a large extent would be reflected back from the boundary. When given the proper mechanical advantage, which produces an increase in the pressure, the transfer of energy is made without loss.

We have studied this action of the middle ear in experimental animals in which exact measurements of the ear's operation may be made in terms of the cochlear potentials.<sup>1</sup> Most of the work has been carried out in the cat, a particularly favorable animal for this

---

From the Princeton Psychological Laboratory. This research was supported in part by the Office of Naval Research, under Contract N6-onr 270, T. O. 3, Project NR 145-322. Permission is granted for reproduction, translation, publication, use and disposal in whole or in part by or for the United States Government.

investigation. We have found that a complete removal of the middle ear, except for the footplate of the stapes, results in a serious loss of sensitivity for all tones. The loss is greatest for the middle frequencies, for which it is about 33 db, and somewhat less for the low and high frequencies, for which it is about 25 db; the over-all average of the loss is 28 db. When we calculate the contribution to the sensitivity that an ideal transformer action should give we obtain a value of 30 db. The observed average of 28 db thus signifies that the middle ear mechanism is remarkably well adapted to its purpose.

In our consideration of this problem we have paid attention to the principle upon which the transformer action is based. From the evidence now at hand there seems little doubt that the mechanical advantage is secured mainly, and perhaps solely, through the difference in areas of tympanic membrane and stapes footplate. The pressure of the transmitted waves is enhanced in proportion to the areal ratio of these surfaces.

What has just been said refers to the principle of the transformer action, yet we are still far from an understanding of the specific manner in which this action is achieved. We need to know more about the acoustical properties of the middle ear.

A mechanical transformer, though simple enough in conception, is a particularly difficult mechanism to construct. Such a transformer ought merely to provide a means for one medium to act upon another, without interposing properties of its own upon the action pattern. No real transformer fills this requirement perfectly; every mechanism has peculiar response characteristics that disturb the action. The difficulties with such a mechanism grow increasingly serious when it is required to operate over a wide frequency range.

No man-made mechanical transformer with which we are acquainted even approximates the quality of the middle ear. A comparable instrument is the early type of phonographic recorder, in which the motions of a diaphragm are communicated to a stylus that inscribes a wave on a wax surface. The poor performance of this instrument is well known. It has become obsolete for this reason, having given way to much more elaborate arrangements of electro-mechanical devices that permit the inclusion of electronic circuits for increasing the sensitivity and correcting for the bad frequency characteristics. Even these modern arrangements do not often have any outstanding qualities of range and fidelity.

As we perceive these engineering difficulties we gain an added appreciation of the instrument that Nature has developed, and of its

fabrication out of such seemingly crude materials as fiber and bone. Our task is to examine further the properties of this instrument.

One of the ways in which the acoustical properties of a system are revealed is through the changes of phase that the system introduces into the vibratory motions that it transmits. The present study represents an attempt to treat the middle ear by this method. As will soon be clear, this method is most profitable when carried out simultaneously with determinations of the sensitivity of the mechanical system.

#### EXPERIMENTAL PROCEDURES

These experiments, like the others already referred to, were performed on the cat, and by using the electrical potentials of the cochlea. The animals were deeply anesthetized with a solution of diallylbarbituric acid injected intraperitoneally, and in nearly all instances they were further immobilized with curare. The curare was injected into the femoral vein in an amount sufficient to suppress all reflex activity, and the animal then was maintained for the duration of the experiment by artificial respiration. The observations were made on 20 ears.

The auditory bulla was approached by the mediolateral route and opened sufficiently to give access to the round window. A platinum foil electrode was placed in contact with the round window membrane and an indifferent electrode was inserted in muscle tissue near by.

The further operative procedures were carried out in two stages. In the first stage the pinna and the membranous part of the external auditory meatus were removed, thereby exposing the drum membrane to clear view. An acoustic probe was inserted into the remaining meatal cavity as near to the drum membrane as was considered safe—within about 2 mm. A sound tube leading from a loudspeaker was placed near the meatal orifice and then this region was sealed over with wax. The sound tube and probe tube thus were enclosed in a small cavity whose inner boundary was the drum membrane.

The acoustic probe consisted of a narrow tube, 1.6 mm in outside diameter, 0.88 mm in inside diameter, and 63.5 mm long. It led to a cap fitted close to the diaphragm of a condenser microphone (Western Electric miniature microphone Type 640-AA). This probe-microphone combination permits the recording of sounds in narrow places, places impossible of access with the microphone itself. The combination, with its associated amplifier, is calibrated to show the sound pressures present at the probe tip.

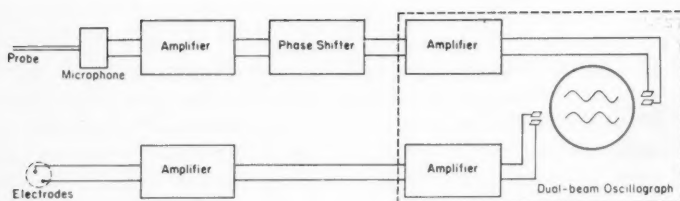


Fig. 1.—Apparatus arrangement for phase measurements.

The acoustic probe and microphone were used also to indicate phase relations. The arrangement for this purpose is shown in Fig. 1. The currents from the microphone amplifier were run through a phase shifter to one side of a dual cathode-ray oscillograph. At the same time, the cochlear potentials picked up from the round window membrane were run through an amplifier to the other side of the dual oscillograph. This oscillograph employs a dual-beam tube: two separate electron streams impinge upon the same tube face and give separate waves that may be placed side by side for comparison.

The microphone and cochlear potential signals were connected to the oscillograph so as to give vertical deflections on its tube face, while the sweep circuit of the instrument gave synchronized horizontal deflections. The same sweep was used for both beams, so that when the two signals were in phase the resulting waves could be made to coincide if suitably adjusted in amplitude and in vertical position on the tube face. When the signals were out of phase the waves were displaced in the lateral dimension with respect to one another. They could be brought into coincidence then only by manipulating the phase shifter in the microphone circuit.

Our procedure at this stage consisted of presenting a tone to the animal's ear and adjusting the phase shifter for coincidence of the stimulating and response waves. The reading of the phase shifter was then recorded. This reading includes the phase characteristics of all the apparatus and of the ear; it has no absolute significance. Such readings were obtained for numerous tones—often as many as 60 to 70—over the range from 100 to 10,000 cycles.

Then the second stage of the operation was carried out. The drum membrane was removed and the incudostapedial joint was broken, after which the malleus and incus were removed. The stapes was left in position in the oval window, with its tendon still attached. Special care was taken to avoid injury to the oval window.

The probe tube was inserted deep in the middle ear cavity, close to the stapes. The sound tube remained in its former position at the meatal orifice, and again the region was sealed over with wax.

Now, with the same tones as used before, the phase shifter again was adjusted for coincidence of the waves representing the sound stimulus and the cochlear response, and its readings noted. A comparison of this series of readings with the series taken earlier reveals the effect of the removal of the middle ear structures upon the phase of the transmitted sound.

Along with these observations of phase we carried out measurements of sensitivity in the usual manner. Using the same tones, both before and after the middle ear operation, we ascertained the sound pressure necessary to produce a standard amount of cochlear response. The differences between these sets of measurements represent the middle ear's contributions to sensitivity.

The observations of phase incur certain technical difficulties. To obtain the necessary stability of the measuring apparatus it is necessary to give special attention to its design and adjustment and to make frequent checks of calibration. Particular difficulties were experienced at 100 cycles. At this frequency our loudspeaker gave only a small output before it suffered overloading as shown by non-linearity and distortion of wave form. Unfortunately also the sensitivity of the cat's ear at this frequency is rather poor. Consequently the signals obtained were small and background noise was disturbing, so that the measurements are not very reliable. At higher frequencies, however, it usually was possible to repeat the measurements within  $5^\circ$ . This remained true after ordinary readjustments of the stimulating arrangement: the wax seal could be removed, the probe tube shifted to a new position, and the seal restored, without altering the readings.

#### RESULTS

Figure 2 shows the phase observations on 4 ears. Each curve represents for a given ear the changes of phase undergone by the transmitted wave due to the presence of the middle ear apparatus. The changes are shown on the ordinate as positive (signifying a retardation of phase) or as negative (signifying an advancement of phase) for various frequencies as given along the abscissa.

The pattern of phase changes is remarkably similar for the different ears, though there are variations of detail. The similarity is especially striking over the lower half of the frequency range, from 150 cycles up to 2000 cycles. Over most of this range, up to 1000 cycles, the middle ear produces a small and fairly uniform advance-

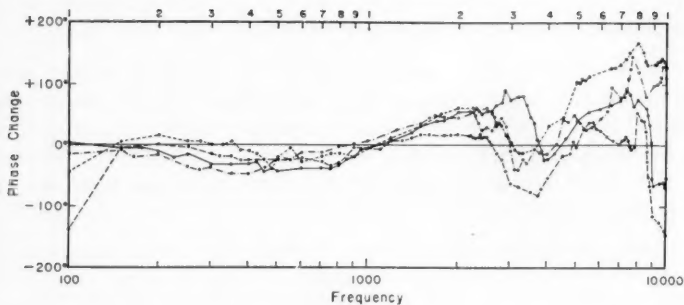


Fig. 2.—Phase changes introduced by the middle ear into the sounds that it transmits. Positive degrees represent phase retardation; negative degrees represent phase advancement.

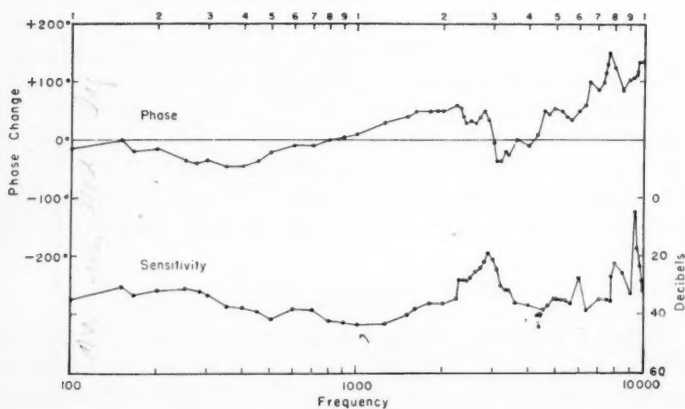


Fig. 3.—Phase changes in the middle ear in relation to its contributions to sensitivity. For the upper curve use the left-hand ordinate scale, and for the lower curve use the right-hand ordinate scale.

ment of phase, never exceeding  $40^\circ$ . Just above 1000 cycles the curves cross the zero line, and here the transmission occurs without phase change. For higher tones the phase change is contrary in direction: the conducted wave lags in phase behind the entering wave. The lag grows in amount to a maximum whose frequency location varies in the different ears from 2000 to 3000 cycles, whereupon the curves fall sharply and cross the zero line again. The points of these crossings vary from 2600 to 3750 cycles. The curves attain a rather sharp minimum, representing the utmost of advancing phase in this region, and then once more rise and cross the zero line. They make this crossing in the region from 3800 to 5000 cycles. In the remaining high-frequency region are the largest variations. Two of the curves remain above the zero line, representing phase lag, and undergo a few undulations there. The other two, likewise undergoing rapid changes, end below the zero line.

These phase changes that the middle ear introduces become more meaningful when viewed along with the changes of sensitivity. The relations are shown for one ear in Fig. 3. Here the upper curve represents phase changes as before and the lower curve represents in decibels the gain in sensitivity afforded by the middle ear.

As we have found earlier, the sensitivity function for the middle ear contains two regions of relatively high sensitivity separated by a sharp peak of low sensitivity. As represented in Fig. 3, the minimum portions of the curves are regions of maximum sensitivity. The first of these regions is very broad; the curve declines slowly with frequency to the region of 1000 cycles and then rises a little more rapidly. Note that this minimum corresponds approximately with the frequency where the phase curve changes from negative to positive. The sharp peak in the sensitivity curve has its maximum (point of poorest sensitivity) near 3000 cycles. This is the frequency where the phase makes a change in the contrary direction, from positive to negative. The second minimum of the curve (maximum of sensitivity) lies between 4000 and 5000 cycles. In this region the curve of phase change again crosses from negative to positive. For still higher frequencies both the sensitivity and phase functions show further variations, but in general the sensitivity is falling while the phase is growing more and more retarded.

#### DISCUSSION

On the basis of the results before us we now proceed in our effort to ascertain the mechanical properties of the middle ear. A mechanical system that only transmits vibrations and does not itself produce them consists of just three types of elements: masses, com-

pliances, and mechanical resistances. The mass of a body represents (among other things) its ability to store energy on being set in motion. Compliance is a property that characterizes a spring: a spring gives way to an applied force, and at the same time exerts a force in opposition. Thus a spring also is able to store energy. Alternatively we speak of a spring as having stiffness, which is the inverse of the compliance. Mechanical resistance is the friction produced by the motion of a body as its parts slide on one another or on fixed structures near by and in so doing use up a part of the applied energy by converting it into heat.

Any analysis of the conductive properties of the middle ear must ultimately be made in the terms just given, but such an analysis is difficult to carry out directly. Rather, we resort to a method of analogies, as is common practice in dealing with complex vibratory systems. In this method we first consider the forms of electric circuits that possess the same phase and sensitivity characteristics as shown by the middle ear, and then by means of analogical relations we translate the electrical properties into mechanical ones. This procedure has the advantage of an extensive fund of experience with electric networks, gained through the work carried out in recent years on various forms of communication systems. On the basis of this experience our insights are richer and better guided than if we dealt with the problems immediately in mechanical terms.

It is important in this connection to point out that the analogies with which we shall deal are not merely connotative, but hold in the strictest sense. This is so because the mathematical expressions relating the variables in one system are identical in form with those in the other system. Hence if we know the form of action for one system we can fully predict it for the analogous system. It may be added further that our actual data consist of electrical measurements, and that to regard them as representing the motions of the air and of the ear we have to rely on certain constant electromechanical relations. In other words, all our acoustic determinations involve functional dependencies between electrical and mechanical systems, and the analogies are only logical developments of these relationships.

Like the mechanical system discussed above, an electrical system that is merely transmissive contains only three types of elements. They are inductances, capacitances, and electrical resistances. Inductance is the property of a coil of wire; it opposes the passage of an electric current both as the current is introduced and as it is withdrawn. Temporarily it stores energy in a magnetic field, an action analogous to the storage of mechanical energy by a mass.

Capacitance is the property of a condenser, which consists of a pair of conducting plates separated by an insulator. When an electric charge is placed on one of the plates a charge of opposite polarity is induced on the other plate and a stress is introduced into the insulator. The condenser thus stores electrical energy much as a spring stores mechanical energy. Electrical resistance is the property of a conductor by virtue of which a passing electric current increases the molecular motions of the substance of the conductor, so that part of the impressed electrical energy is converted into heat.

The kind of opposition to the flow of alternating current that an inductance causes is known as positive reactance; its amount increases progressively with the frequency. The kind of opposition that a condenser causes is known as negative reactance, and its amount varies inversely as the frequency. It is also characteristic of an inductance to retard or lag the phase of the alternating current passing through it, whereas a capacitance advances the phase of the conducted current.

In our analogical system, mass corresponds to inductance, compliance to capacitance, and mechanical resistance to electrical resistance.

Now let us regard the results of Fig. 2 and 3 as representing the behavior of an electric network, and consider what arrangement of inductances, capacitances, and resistances would produce such effects in response to different vibratory frequencies. To simplify the problem somewhat at this point we consider the curves only over the principal portion of the range, neglecting the variations around 100 cycles and above 8000 cycles where experimental conditions and individual differences present particular difficulties.

In the low-frequency region, up to about 1000 cycles, the function shows a negative phase change. Imposed currents with frequencies within this range are advanced in phase. The representative electric network therefore must be one in which capacitative reactance is predominant within this frequency range. Above 1000 cycles and nearly to 3000 cycles, however, the phase change is positive or lagging, which means that for this range the inductive reactance of the network must be predominant. Then above 3000 cycles, as the phase change again becomes negative, the capacitative reactance again is predominant, and, finally, for the upper frequencies the phase change is positive and inductive reactance once more is predominant. At those frequencies at which the function crosses the zero phase line the inductive and capacitative effects are balanced

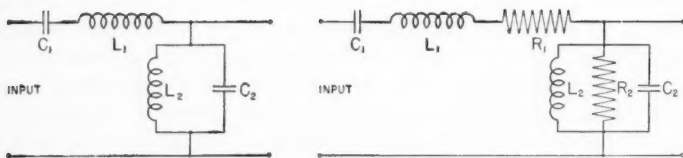


Fig. 4.—Electric networks presented as “models” of the middle ear mechanism. Circuit *a*, on the left, contains only inductances ( $L_1$ ,  $L_2$ ), and capacitances ( $C_1$ ,  $C_2$ ), whereas circuit *b*, on the right, includes resistances ( $R_1$ ,  $R_2$ ) also.

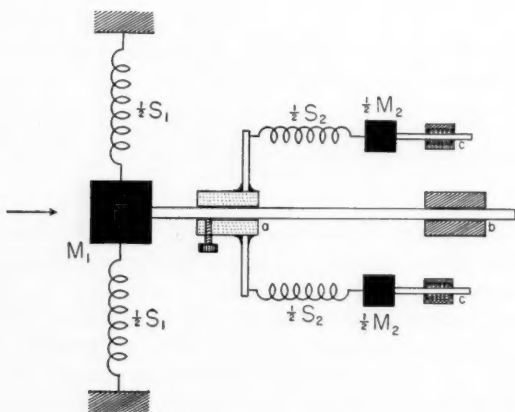


Fig. 5.—The mechanical counterpart of Fig. 4. With the proper conceptual changes this diagram will serve as the analogy for either circuit *a* or circuit *b* of that figure.  $M_1$  and  $M_2$  are masses,  $S_1$  and  $S_2$  are springs, *a* is a link, *b* and *c* are bearings. The cross-hatched parts are immobile.

For one condition, when serving as the analogy of circuit *4a*, link *a* is rigidly attached to the central shaft by means of the thumb screw and bearing *b* is frictionless. For the other condition, when serving as the analogy of circuit *4b*, bearing *b* is assumed to contain friction  $F_1$ , and the thumb screw of link *a* has been loosened and friction  $F_2$  in this link governs the transfer of motion to  $S_2$  and  $M_2$ . Under both conditions, bearings *c* are frictionless, and all shafts and link *a* are massless.

and their net reactance is zero. When the crossing is upward, and negative reactance gives way to positive reactance, we have a point of resonance, and when the crossing is in the contrary direction, and positive reactance gives way to negative reactance, we have a point of antiresonance. There are two resonance points, one around 1000 cycles and another varying in location in the different curves between 3800 and 5000 cycles, and interposed between them is an antiresonance, varying in location from 2600 to 3750 cycles.

As an approach to the disclosure of a responsive system with the properties just described let us first consider a relatively simple electric circuit, one containing four circuit elements, two inductances and two capacitances. There are four ways in which these elements may be arranged, and the arrangements are electrically equivalent if the elements are given the proper values. One arrangement is shown in Fig. 4*a*. It consists of a capacitance  $C_1$  and an inductance  $L_1$  in series, followed by a capacitance  $C_2$  and an inductance  $L_2$  in parallel. The reactance of this circuit varies with the frequency in the following manner. At zero frequency the reactance is negative, and infinitely large. As the frequency rises this negative reactance grows less, at first rapidly and then more slowly, until it reaches zero at a frequency  $f_1$ , the first resonance frequency. Above  $f_1$  the reactance is positive, and rises precipitously to infinity as the frequency approaches  $f_2$ , an antiresonance frequency. At  $f_2$  the function becomes discontinuous, suddenly vanishing at positive infinity and reappearing at negative infinity. Above  $f_2$  the negative reactance progressively grows smaller until it is zero at  $f_3$ , the second resonance frequency. Above  $f_3$  it is positive, and rather gradually approaches positive infinity as the frequency becomes infinitely high.

This function has the desired form except for the wide range of variations and the discontinuity at the point of antiresonance. However, these undesirable features vanish under practical conditions. In hearing we are not concerned with frequencies near zero and infinity. Also, real circuits always contain resistances along with the other elements, and in the presence of resistance there are no discontinuities in the function. Instead of jumping from infinitely positive to infinitely negative reactance at the antiresonance frequency, the function simply falls more or less rapidly from positive to negative through zero.

The introduction of resistances into the suggested circuit is shown in Fig. 4*b*. These resistances have further desirable effects. They greatly reduce the amount of the phase change introduced into the transmitted signal. At the same time they reduce the mag-

nitude of the peaks of response at resonance frequencies and of the diminution of response at antiresonance frequencies. From Fig. 2 and 3 it is evident that a high value of resistance is effective about the first resonance point  $f_1$  and a somewhat smaller value about the antiresonance and second resonance points  $f_2$  and  $f_3$ . This we infer because in the lower frequencies the variations of phase are small and the resonance curve is very broad, whereas in the higher frequencies the phase variations are larger and the response more rapidly changing. The antiresonance, shown best in Fig. 3, is particularly abrupt.

We are now ready to turn back to the ear and to translate our electrical conceptions into the mechanical form. If we consider first the analogy to the simpler circuit of Fig. 4*a* we shall have a mechanical system consisting of two masses and two springs (compliances). The first mass  $M_1$  is acted upon directly by the applied force, and is attached to the spring  $S_1$  in such a way that when it is moved away from its equilibrium position the spring tends to bring it back. The second mass  $M_2$  is not acted upon directly by the force, but is attached to the first mass through a spring  $S_2$ . This mechanism is pictured in Fig. 5, where the black squares are masses, the coils are springs, and certain shafts and bearings are provided to link the elements together. (In the drawing the springs  $S_1$  and  $S_2$  and the mass  $M_2$  have been split into symmetrical halves in order to present an appearance of dynamical balance; this arrangement makes no difference otherwise.) At this point we are to suppose that the linkage at  $a$  is a rigid one, having been made so by means of the thumb screw, and the bearings  $b, c$  are frictionless. Also, the link  $a$  and all the shafts are to be considered massless.

This same diagram will serve to represent the mechanical counterpart of Fig. 4*b*, if now we think of bearing  $b$  as containing friction and the thumb screw of link  $a$  as having been loosened so that the coupling between central and side shafts depends upon friction. The friction  $F_1$  in bearing  $b$  uses up some of the energy communicated to the mass  $M_1$ , and the friction  $F_2$  in link  $a$  uses up some of the energy that would otherwise be transferred to  $S_2$  and  $M_2$ .

Our final consideration is the possibility of identifying the elements  $M_1, M_2, S_1, S_2, F_1$ , and  $F_2$  in terms of anatomical structures in the ear. We present the following as a reasonable hypothesis.

The mass  $M_1$  is the mass of the malleus, incus, and drum membrane, and other parts that move strictly in phase with them. The spring  $S_1$  is partly in the drum membrane and partly in the suspension system of the middle ear. The spring  $S_2$  is wholly in the suspension system: in the attachments afforded by the anterior and

lateral ligaments of the malleus and its wing process, the posterior ligament of the incus, and the tensor tympani tendon. The mass  $M_2$  is partly in this suspension system and partly in the structures to which the suspending elements are moored. These mooring structures add to the mass because they are not altogether rigid, but are somewhat involved in the motions. The friction  $F_1$  is the friction generated by the movements of mass  $M_1$ . It includes the sliding of fibers of the drum membrane over one another, which occurs especially near its outer edge where the central motion grades down to zero under the restraint afforded by the tympanic ring. It includes also a part of the friction produced in the ligaments and the tensor tympani tendon, and especially that produced in their more proximal portions. The friction  $F_2$  is generated in the movements of mass  $M_2$ , and it arises in the more distal portions of the suspensory elements as their fibers move on one another or over fixed structures.

#### CONCLUSIONS

Our observations have been limited to the cat, and necessarily our conclusions have direct application only to this species. At the same time, however, the possibility is presented to us that other ears, including man's, have made adaptations of function that are as serviceable as the ones revealed in this animal.

We find that the middle ear apparatus—or, rather, that part of it peripheral to the stapes—fulfills its duties as a mechanical transformer with only slight disturbances of the response pattern. It presents to the stimulating sounds an impedance that varies remarkably little with frequency. It does this because it is a doubletuned and highly damped system. To the single stiffness and single mass that would have to be present in order for the structure to act as a mechanical transformer have been added a further stiffness and mass, with the result that we have a system resonant to two separated frequencies. At the same time, the action is subjected to considerable frictional damping, so that the resonances are very broad.

We have so far left out of account the behavior of the system at the extreme frequencies, for the reasons given. The results at the lowest frequencies suggest that the response continues to be more uniform at this end of the scale than we ought to expect from a system containing only four reactive elements (two masses and two compliances). If further investigation supports this suggestion it will be necessary to add one more reactive element to our representative scheme.

We believe that the variations found at the upper frequencies represent inherent instabilities in the mechanism: variations in the

tensions of the suspensory structures, and perhaps also in the effective mass which they present. Especially must we expect a variation in the tension exerted by the tensor tympani tendon, even though under our conditions the action of its muscle was largely prevented by curarization.

The part of the middle ear apparatus that has been excluded from this discussion, the stapes and its tendon, will be considered later by a somewhat different method. It suffices to say now that over the major portion of the auditory range this part has only a very slight effect upon the action of the system. That action, as we have seen, commands our respect as an achievement in mechanical engineering and in the service of hearing.

#### SUMMARY

The characteristics of the middle ear apparatus have been investigated in terms of the changes of phase and magnitude that this apparatus introduces into the sounds that it transmits. The work has been carried out on the cat by means of the cochlear potentials. The results show that the middle ear in this animal presents remarkably uniform properties to sounds over a wide range of frequencies. An analysis based upon electrical and mechanical "models" indicates that this uniformity is achieved by the development of two mechanical resonances separated by about two octaves and by the presence of a high degree of frictional damping. In consequence, the middle ear is able to carry out its function as a mechanical transformer with minimum disturbance of the response pattern.

We are indebted to the Bell Telephone Laboratories and the Western Electric Company for the loan of calibrated microphones used in this research.

PRINCETON UNIVERSITY.

#### REFERENCE

1. Wever, E. G., Lawrence, M., and Smith, K. R.: The Middle Ear in Sound Conduction, *Arch. Otolaryng.* 48:19-35, 1948.

## II

# STUDIES ON THE HISTOGENESIS OF THE AMPULLARY CUPULA

THURE VILSTRUP

COPENHAGEN, DENMARK

Investigators of the labyrinth generally agree that the exchange of energy between endolymphatic motion and stimulation of the sensory cells of the crista takes place with the ampullary cupula as intermediate agent. The cupula is a small, movable, jelly-like structure, riding on the top of the crista.

A thorough understanding of the structure of the cupula, therefore, is absolutely essential to the interpretation and understanding of the more detailed character of the ampullary or cupular reflexes.

As yet a regrettable lack of knowledge prevails concerning the nature of this structure and, in particular, its connection with the sensory epithelium. This is apparent from the uncertainty concerning the interpretation of a number of physiological observations with regard to the ampullary reflexes.

Therefore it is important with reference to the physiology of the labyrinth to elucidate as far as possible all the available data concerning the histogenesis and finer structure of the cupula. In this paper the author will present the results of his own studies on the histogenesis of this organ, while the results of studies on the finished structure of the cupula will be reported in a subsequent paper.

The intimate relationship commonly observed between the histogenesis of an organ and its structure in the finished state, together with its way of function is found here, too; and the knowledge of the histogenesis of the organ supplements and elucidates the principal features and details in the structure of the finished organ. As the histogenesis and the finished structure are two sides of the same thing, it may be difficult to decide which details properly belong in this paper and which ones should rather be dealt with in the following paper. Some details will therefore be left out here.

---

From the Institute of Histology at the University of Lund, Sweden, and the Department of Oto-Rhino-Laryngology at the University Clinic of Lund, Sweden.

*Previous Investigations.* The studies previously reported on the histogenesis of the cupula itself are rather scanty. Most histogenetic works on sensory epithelia of the labyrinth have been carried out on maculae, and most authors have a priori drawn conclusions from the findings here, about the conditions of the ampullary contents.

Those studies were performed on fetuses of widely different species, and it appears to have been assumed that general conclusions could be drawn from such studies because the ampullae and their contents largely show the same construction throughout the kingdom of vertebrates.

In chicken fetuses (six days after artificial fertilization of the eggs) Herzog<sup>4</sup> in 1925 found a commencing macula-crista differentiation, and he states that at this juncture the sensory epithelia are covered by a net of stainable gelatinous strands that are seen to be connected with the epithelium by granular and filamentous threads. He thinks that there must be an intimate connection between the epithelium and the threads, which he interprets as secretory products of epithelial cells.

Fell<sup>3</sup> in 1928, studying otocysts extirpated from chicken fetuses (cultivated further in vitro), found in the 9-day embryo some distinct and separate labyrinthine sensory epithelia provided with cilia.

The question as to which cells form the cuticular substance is still unsettled. Studnicka<sup>11, 12</sup> thinks it is the hair cells. Kolmer<sup>7</sup> assigns this function to the supporting cells, whereas v. d. Stricht,<sup>9, 10</sup> Wittmaack<sup>18</sup> and Werner<sup>16, 17</sup> think that the cupula develops through co-operation of different elements of the sensory epithelium: hair cells, sensory cells, and intercellular substance.

Nor is it agreed in what way the network of the cupula is formed, as the cupula sometimes is designated as a secretory product, sometimes as an exoplasmic product. The latter view is advocated by Wittmaack<sup>19</sup> who simply asserts that the cupula is made up of an extracellular part of the sensory cells themselves.

Farkas<sup>2</sup> advances an altogether divergent theory, claiming that the cupula is a secretory product, formed layer by layer from the cells of the planum semilunatum.

These and other divergent views indicate the necessity of further investigation into this matter on suitable fetuses and the desirability of additional results that are more clear-cut histologically.

Modern methods of histological investigation afford, in many fields of study, opportunities to draw conclusions which were not

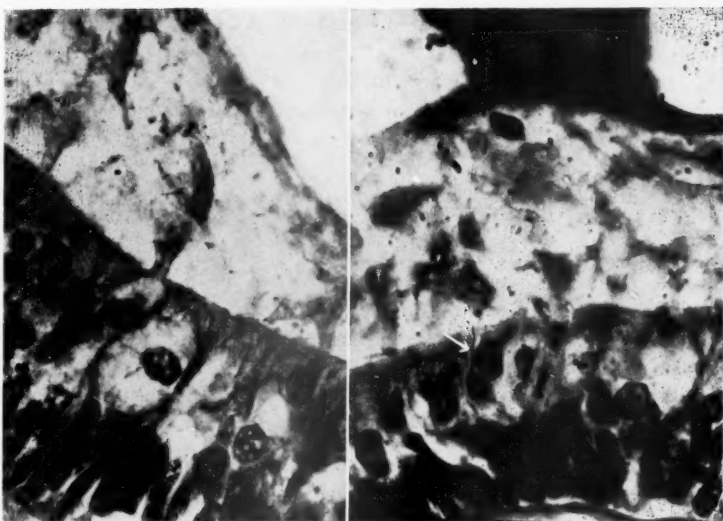


Fig. 1.—Photomicrograph of section through the ampulla (crista) lateralis of a 17-cm shark fetus. Note the cytoplasmic columns and their transition into the cells. (Hematoxylin-eosin stain;  $\times 1000$ .)

Fig. 2.—Photomicrograph of section through fetal shark crista. Note the two cupular zones and the protrusion from a luminal nucleus (indicated by the arrow). (Toluidine blue stain;  $\times 250$ .)

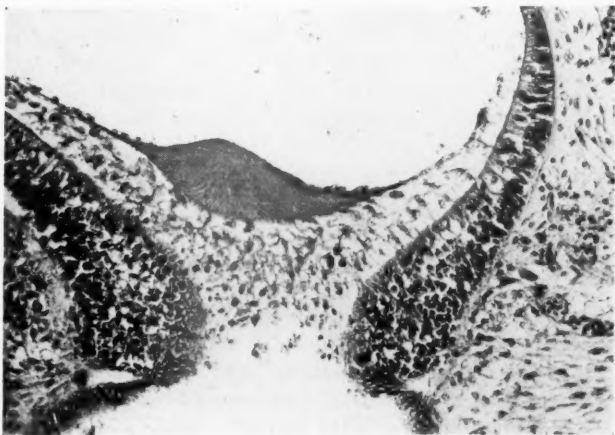


Fig. 3.—Photomicrograph of section through lateral crista, cut obliquely. Note the two cupular zones and the extent of the net-like subcupular zone. (Fig. 1 shows an area of this section under high power.) (Hematoxylin-Best's carmine stain;  $\times 100$ .)

available to earlier investigators, and it is partly on such methods that the work in this paper was based.

#### HISTOLOGICAL INVESTIGATION

*Material.* The material for the studies here reported comprises 10 shark fetuses. All the fetuses were removed from living female sharks (*Acanthias vulgaris*), and all the fetuses were living and moving at the time of the fixation, which took place immediately after their removal from the mother. The smallest fetuses studied here (measuring less than 5 cm in length) originate from material in the collections of the Törnblad Institute in Lund, Sweden, and detailed data have not been available concerning their fixation. All the other fetuses (my material) had their belly and cranial cavity opened in the midline, and were at once placed in toto in formalin-mercuric chloride solution, and after 3 weeks transferred to formalin-alcohol. The two incisions—in the belly and into the cranial cavity—did not kill the animals.

The further treatment of these specimens took place after an additional 2-3 weeks, but a few of them were left in formalin-alcohol for up to 5 weeks. Control experiments showed that fixation of the cranial contents of these fetuses took place quite instantaneously when the cranial cavity was opened beforehand.

After fixation was completed the heads were cut off and divided by transversal section immediately in front of the labyrinths,\* so that the specimen blocks essentially consisted of the two labyrinths. The blocks were embedded in paraffin (56° C.) and, with one exception, cut in the transversal plane into sections of 10 $\mu$ . Beforehand, the blocks were adjusted in relation to the microtome knife so that the right and left labyrinths were hit by the knife at the same time in the same place and thus were comparable in the sections.

Some of the sections were placed on especially cleaned slides and submitted to micro-incineration; others were deparaffinized in xylol and examined directly under the phase contrast microscope (Zeiss), without being stained. The rest of the sections were stained respectively with hematoxylin-eosin, chromotropic hematoxylin and gallocyenin-chrome alum (Einarson,<sup>1</sup> 1932). Some sections were stained after the Feulgen method, and most of the remaining sections were stained with toluidine blue (pH 5.5), among other reasons because this compound stains the cupular fibrils particularly well.

---

\*In 6 cases the snout was cut off immediately before fixation.

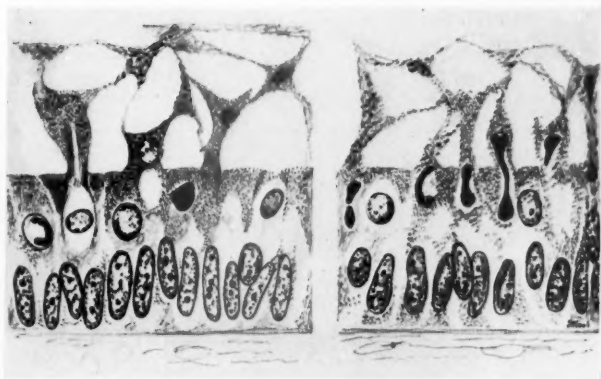


Fig. 4.—Schematic drawing of stages observed in the migration of cytoplasm from the crista. To the left, cytoplasmic columns; to the right, stages in the migration of the nucleus. Note the chromatin pattern: first a "string of beads" beneath the nuclear membrane, then the "horseshoe formation," and then the "peg formation."

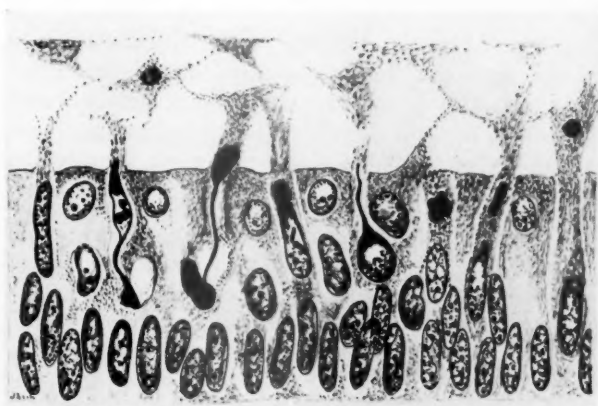


Fig. 5.—Schematic drawing of different stages observed in the differentiation of the chromatin, chiefly in the cells of the basal nuclear layer. To the right a cytoplasmic column from a basal cell is seen. Photomicrographs of these stages are shown in Fig. 15, 16, 17 and 18.

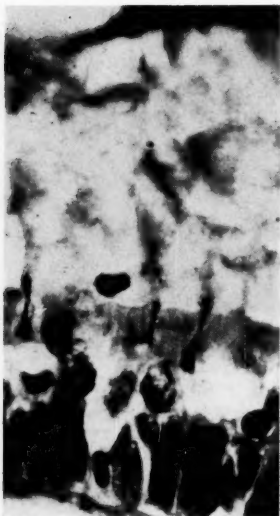


Fig. 6.—Photomicrograph showing, *above*, a corner of the fibrillary cupular zone; *below*, the subcupular zone. Note the cytoplasmic columns and their "pegs." (Toluidine blue stain;  $\times 630$ .)

While after fixation and drying by the Altmann-Gersh method, toluidine blue gives these fibrils a beautiful metachromatic (reddish-violet) stain signifying the presence of mucopolysaccharides, the cupulae in adult animals as well as in the fetuses take most often a purely blue stain after chemical fixation (while the macularia after the above-mentioned fixation take a metachromatic stain).

The Feulgen reaction\* and the gallocyanin-chrome-alum staining gave particular information about the nuclei of the cells and, what in this connection is of special interest, about the nature of the nucleochromatin-like extranuclear formations. Staining with toluidine blue made the cytoplasmic extensions more conspicuous, which, among other things, made the sections more suitable for photography.

*Findings.* The development that will be outlined in the following was seen (in a single case) to have commenced in shark fetuses of about 7 cm in length. It may be followed even in the largest fetuses here examined, i.e., animals measuring about 19 cm in length. Evidence of the same processes as are described may also be seen in

---

\*It stains nuclear chromatin (desoxyribonucleic acid) electively.

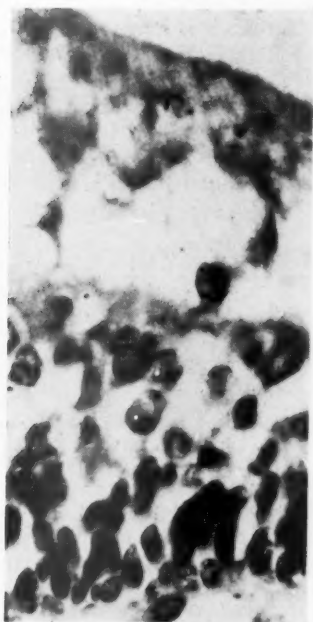


Fig. 7.—Photomicrograph showing emigrating, nonpyknotic nucleus. (The structure does not stand out well.) (Hematoxylin-eosin stain; x630.)

the ampullae of the adult cod, signifying processes of maintenance and reparation similar in character to the processes of development proper.

In the earliest stages of the shark fetus, under 7 cm, merely thickening of the epithelium is seen at the places of the maculae and cristae. Here the cells within the areas of sensory epithelium show no evidence of differentiation, and there is no sign of the presence of gelatinous substance.

After this, cuticular substance develops first on the sensory epithelia in the inferior part of the labyrinth (macula sacculi, papilla neglectae and macula lagenae). Then gelatinous substance appears on the macula utriculi, and not until now do the characteristic cellular processes, which will be mentioned further on, appear in the cristae. It is interesting to see that the dorsolateral tongue-like extension from the macula utriculi (lacinia) develops its gelatinous

substance somewhat later than the rest of this macula, so that in sections from larger fetuses it is seen together with the crista lateralis and at the same developmental stage as this structure.

In this paper only the developmental aspects of the cristae will be mentioned, and introductorily it may be established that the differentiation of the three cristae appears to take place at about the same time, and that the cellular processes take a quite uniform course in the three cristae, as far as may be observed.

At the stage where the building up of the cupula commences the sensory epithelium is seen to be made up of cells with their nuclei situated in two layers (Fig. 1). Basally, long, cigar-shaped, or slightly curving, nuclei are seen, with pronounced chromatin configuration, and with the chromatin distributed in close-packed granules within the nuclear membrane. Nucleoli cannot be made out with certainty. Luminally, at the place of the sensory cells, we find goblet-shaped cells with mediobasal spherical nuclei, in which the chromatin also appears distributed in fine granules. On the whole, these nuclei are a little lighter in color than the basal nuclei. From the basal cells the cytoplasm extends in the form of thin processes up towards the lumen and in between the luminal cells.

This form of structure quite corresponds to the findings in the adult shark, in which the two layers of cells were seen clearly, and in which the nuclei are situated most regularly, as two rows of pearls on parallel strings.

In the fetuses this regularity is less pronounced. Here the basal nuclei are often seen to have pushed forward, towards the lumen, and occupying a space in the light zone between the two rows of nuclei, or even having made their way up to the luminal border of the epithelium (Fig. 13 and 14). Other factors contributing to disturbance of the regularity will be mentioned later.

The microscopic picture of the fetal crista shows long and broad columns of cytoplasm extending from the crista epithelium out into the lumen, where they enter into the formation of a network (Fig. 1, 3, 4 and 5). Large chromatin bodies or even nuclei are seen to push out into these columns (Fig. 4, 5, 11, 12 and 17), so that the zone at the level of the crista is characterized by the presence of the cytoplasm and nuclei or nuclear material (Fig. 3, 4 and 5). This statement is based upon the observation of every sensory cell in all the cristae and maculae in the 10 fetuses examined. What is seen in each individual section— or in each individual cell— is merely a snapshot. Through the observation of a great number of such snapshots it is possible to put the details together and thus

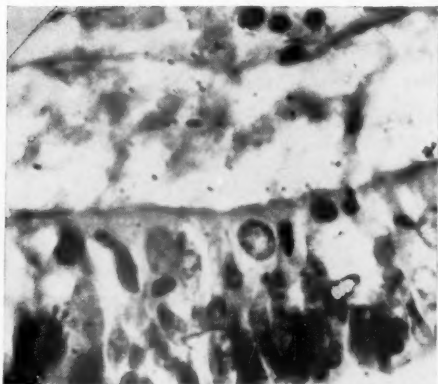


Fig. 8.—Photomicrograph showing part of the subcupular zone with chromatin masses. In the cytoplasmic column, two chromatin formations are seen, one of them intra-epithelially. (Toluidine blue stain; x630.)

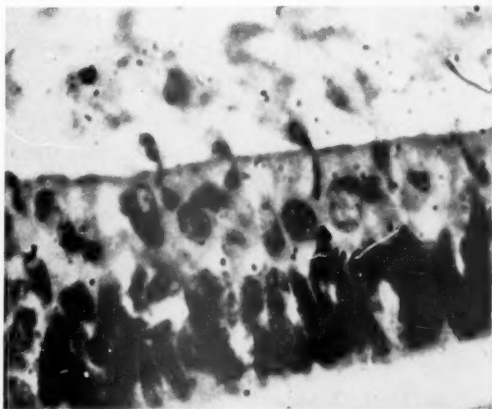


Fig. 9.—Photomicrograph showing emigrating chromatin "pegs," partly dumb-bell-shaped. (Toluidine blue stain; x630.)

obtain a "film." It is to be emphasized, however, that even though each snapshot described in the following has been ascertained unquestionably in a great many cases, the composition of the individual pictures naturally will be subjective, thus including possibilities of error. In order to reduce the frequency of such errors as far as possible, the author has examined several fetuses in the different size groups, and every individual crista-macula cell in each section. The conclusion of this work is as follows:

First, a column of cytoplasm is projecting out into the lumen, for instance, from a luminal crista cell (Fig. 1). The width of this column at its site of origin corresponds to from one-half to three-fourths of the width of the cell. The column grows lengthwise and may become several times longer than its mother cell. As a rule, the finished column is broadest in its luminal part, thus becoming conical, with its narrow base in the mother cell. Not infrequently the lighter central cytoplasm of the mother cell is seen to extend out through the central part of the luminal process (Fig. 1), and this may perhaps signify that the column is canalized. The nucleus of the mother cell, then, undergoes "transformation" or dedifferentiation into a peg-shaped or dumb-bell-shaped body of nuclear chromatin (see below), perpendicular to the luminal borderline of the epithelium, and migrates out into the preformed cytoplasmic column. All sorts of transitions are found between entirely intra-epithelial "pegs," pegs halfway intra-epithelial and half-way intra-luminal, and pegs entirely extra-epithelial, at any distance from the epithelium.

Most often the "peg" is of dumb-bell shape, that is, consisting of two spherical bodies connected by a rod-shaped intermediate part. Not infrequently, the latter structure is defective or "broken," so that the peg is represented by two spherical bodies. Now and then these balls apparently have moved away from each other, as seen in Fig. 8, one ball located intra-epithelially while the other lies far out in the lumen. That they belong together is evident from their location in the same cytoplasmic column.

These "pegs" appear always to consist of pyknotic nuclear mass, but in not a few cases the luminal end of shorter or longer cytoplasmic columns show nuclear structures which greatly resemble the nuclei that may be seen luminally in the sensory epithelium. Such nuclei present a finely granulated chromatin structure, while the cytoplasm as a rule is stellate and enters into the reticulum formation that is established by all the columns and the cells situated in the lumen (Fig. 5).

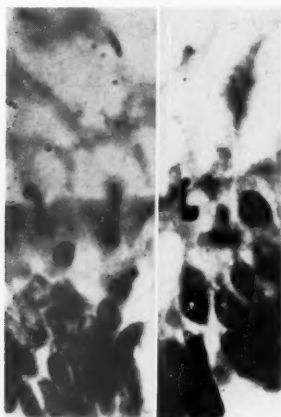


Fig. 10.—Photomicrograph showing an emigrating chromatin "peg" still partly at the "horseshoe stage." (Hematoxylin-chromotrope stain; x700.)



Fig. 11.—Photomicrograph showing a chromatin "peg" in its column. (Toluidine blue stain; x630.)

Fig. 12.—Photomicrograph showing emigrating chromatin "pegs" in their columns. (Toluidine blue stain; x800.)

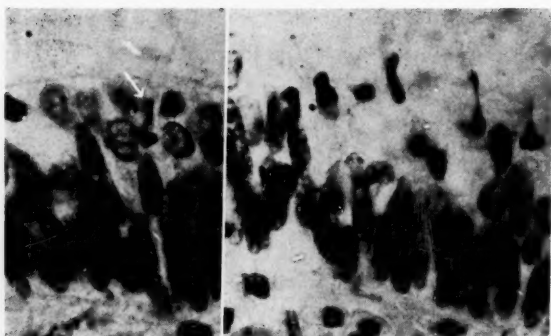


Fig. 13.—Photomicrograph showing emigration of chromatin "pegs." The arrow points at a stage of the chromatin differentiation. (Feulgen stain;  $\times 620$ .)

It is rather interesting to establish that from the specimens it is clearly evident that the basal cells as well as the luminal cells enter into the formation of this luminal reticulum.

Often a basal cell is seen to project its cytoplasm as a wide bridge between two luminal cells, and this bridge continues unbroken out into the lumen, forming a column of quite the same appearance as mentioned above. The intra-epithelial part of such cytoplasmic structures is difficult to photograph and cannot be made as plain in the pictures as would be desirable, whereas it is easy to follow in the sections (Fig. 5).

Nuclear material is then pushed out in these columns, but this is usually preceded by a short migration of the nucleus of the basal mother cell, which is pushed in front of its neighbors, breaking thus the continuity of the row of cells (Fig. 14).

The more precise character of these most peculiar nuclear migrations cannot be established with certainty.

According to his observations, the author thinks that this process conceivably may commence in two ways. Either the initial step is a mitosis (and mitoses are of frequent occurrence in both layers of the sensory epithelium at these stages), and one daughter nucleus (or cell) then emigrates from the sensory epithelium, or the process commences in an entirely different way.

Most findings suggest that a part of the nuclear chromatin collects at a suitable place in the nucleus before it is pushed out of this



Fig. 14.—Photomicrograph showing to the right a cigar-shaped emigrating nucleus from the basal layer. Its luminal part shows absence of structures, while the basal part of the nucleus has preserved its structures. (Feulgen stain;  $\times 630$ .)

under the formation of a rod- or club-shaped structure (Fig. 4). At this introductory stage, in the luminal nuclei, the nuclear chromatin collects as small granules forming a long "string of beads" in a horseshoe (Fig. 10) or ring shape. The string of beads is situated immediately beneath the nuclear membrane. At the next stage one end of the horseshoe is pushed out of the nucleus, while the rest of the chain follows slowly. As a characteristic feature, in these nuclei all the chromatin appears to enter into the formation of this chain. Still, it cannot be decided quite conclusively whether the entire "horseshoe" is pushed out of the nucleus, even though this is more likely. Nor can it be observed that this process is introduced by opening of the nuclear membrane. As a matter of fact, no such opening can be made out with certainty throughout the entire process.

The next observable stage is the one shown in Fig. 4 and 10, in which the chromatin forms a pyknotic amorphous horseshoe-shaped "peg." As to the basal nuclei, most observations indicate that the development here proceeds in a slightly different way. For here, especially in Feulgen-stained sections, the luminal part of the protruding basal nucleus becomes structureless, appearing quite black. No ring formation is seen here. Then the luminal part of the nucleus is pushed forward, towards the lumen, and in some cases it may be seen far away from the mother nucleus, though still connected with this by a long stem, which in Feulgen specimens stains in quite the same way as the mother nucleus. Figure 18 shows such a projected part of the basal nucleus situated in or near the lumen,

while the columnar mother nucleus is situated in the luminally placed layer of the sensory epithelium. Similar stalky bodies from luminal nuclei are seen in Fig. 2, 15, 16 and 17. The introductory migration of the basal nuclei need not be accompanied by differentiation of the chromatin—this appears at a later stage.

It is to be mentioned that the crista epithelia in a high degree are characterized by a large number of dark, structureless nuclei of ordinary nuclear size in the luminal layer. Not infrequently these nuclei are seen to be connected to peg-shaped extensions of chromatin (Fig. 9), but this is far from being a general rule. The writer is unable to explain which role these nuclei may play, but it seems reasonable to assume that they represent stages in the above-mentioned process of nuclear differentiation and migration. Similar pyknotic structures comprising the entire nucleus are hardly ever seen in the basal nuclear layer.

As to the cytoplasmic network in the lumen, in the form described here it extends from the outermost sensory epithelial cell on one side of the crista to the most external corresponding cell on the other side of the crista. Quite near the lumen this loose layer is intimately connected with the fibrillary cupula itself. In all sections this structure is stamped by the shrinking effect of the chemical agents, especially in comparison with the cupula in a fresh, frozen-dried ampulla. The fibrillary structure is plainly observable, and in this section of the cupula the fibrils are chiefly parallel, running perpendicular to the free borderline of the sensory epithelium towards the lumen. As will be pointed out in more detail in the following paper, the shrinkage of the cupula may even be noticed by studying the individual fibrils. Under high power, especially with polarized light, the individual fibril is seen to have contracted into the shape of a corkscrew. So, in specimens that have been fixed chemically, there is not much chance of studying the connection between the zone mentioned and the fibrillary section of the cupula. Still, it can be seen distinctly that the marginal fibrils in the shrunken cupula (shrunken longitudinally and also, though in a lesser degree, transversally) are gathered into broad bundles, inserting through long cytoplasmic columns in the marginal crista cells in both cell layers of the crista. Likewise, many of the cytoplasmic columns mentioned may be seen to enter directly into the streaks of the fibrillary cupula.

It is further noticed that also the fibrillary section of the cupula seems to contain a few nuclei, as a rule of the aforementioned "pyknotic" type.

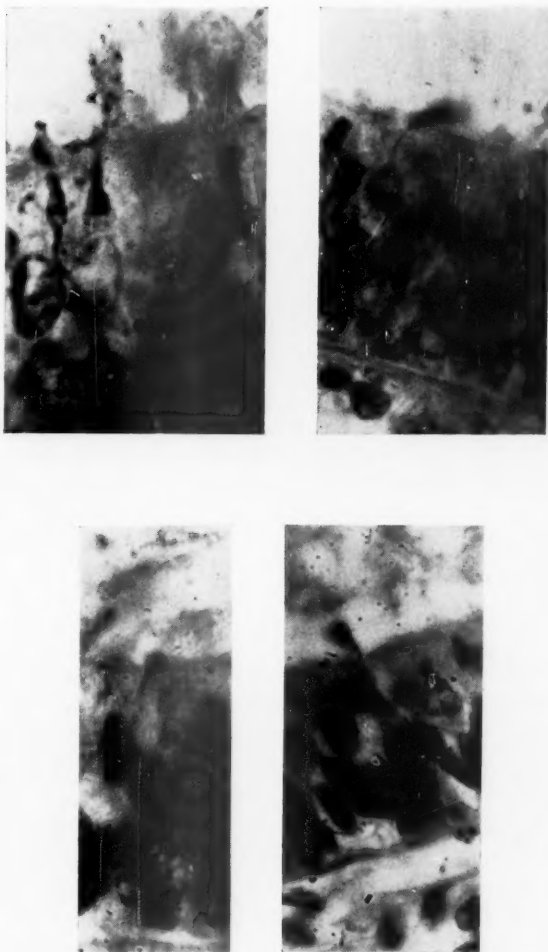


Fig. 15, 16, 17 and 18.—Photomicrographs showing stages in the process of nuclear differentiation, probably in every case arising from the basal nuclei. Here, as in the other photomicrographs, it was necessary to focus sharply on certain details, and thus the other features have become less distinct. All these sections were stained with toluidine blue. The magnification is 1600 in Fig. 15, 1000 in Fig. 16, 1600 in Fig. 17, and 800 in Fig. 18.

Also in the cupula of the adult animals (cod) it is possible after freezing-drying of the specimen to see such cells with hazy pyknotic nuclear remnants and a wide cytoplasmic margin, stellate in outline, situated in the fibrillary cupula.

These cells, which are rather scanty in the frozen-dried cupula, are seen to be provided with extensions that anastomose with extensions from similar neighbor cells, and many of these extensions continue into the cupular fibrils. After this, it seems reasonable to assume that the fibrillary layer is built up from the first-mentioned "subcupular" layer; and it seems obvious to think that this building-up is due to the cells that emigrated from the crista epithelium. Owing to the fixation, the writer has not been able to gather sufficient details to form a clear picture of these processes based on many photomicrographs. From the present observations, however, it appears as if the emigrated cells form a protoplasmic reticulum in the border layer facing the peripheral part of the cupula. This can be observed directly in many sections. Probably this reticulum shifts slowly towards the periphery under its formation, and during this shift the network is submitted to the influence of forces, presumably entirely mechanical, that tend to favor such an arrangement of the reticulum as corresponds to that course of the fibrils which actually is found in the fibrillary cupula. When, finally, the cells are situated in the peripheral marginal zones of the cupula, their task is at an end: the nuclei disappear or are seen merely as shadowy remnants whereas the cytoplasm preserves its stellate form with extensions for a while yet. This picture may be seen in the periphery of the cupula in the adult animal, while the few cells noticeable in the center are greatly diminished in number, but with a little more stainable nuclei.

In this connection it should be remembered that the actual course of the fibrils in the cupula ampullaris is not quite in keeping with the description of cupulae that have been fixed chemically. These cupulae have been so affected by chemical agents that not only have the individual fibrils changed in appearance but the entire fibrillary arrangement, which is exceedingly sensitive and may readily be altered, for instance, by deflection of the cupula, has undergone considerable alteration.

The pyknotic nuclear masses, which in the form of dumb-bell-like formations are pushed out in the cytoplasmic columns, represent a particular problem.

It is difficult to imagine that the crista cells would expel such a large part of their vital nuclear chromatin (see below), if this did

not serve some definite purpose elsewhere. Thus it is unreasonable to assume that the expelled material is to be looked upon merely as waste material removed by expulsion. To the author, therefore, it seems more likely that these nuclear masses serve some purpose in the active building-up process. This problem may perhaps be elucidated by considering the large number of pyknotic nuclei in the luminal layer of the crista (together with the other nuclei, sharply defined and well fixed). These nuclei, too, I think, serve a definite purpose. In spite of various attempts, however, it has not been practicable to enter sufficiently into this question. Still, it seems obvious to assume that the pyknosis plays a part—somehow a conditional part—in the process of nuclear emigration, and that the nuclear mass after the emigration still is able, in connection with the cytoplasm, to perform expedient tasks in the building-up process.<sup>6</sup> Apparently these tasks consist chiefly in the building of the cupular network as described above.

When the writer first found the cellular and, especially, the nuclear processes here described in his sections, the pictures seemed so improbable and so remote from all cellular processes hitherto recognized and described, that *a priori* it seemed more reasonable to refute these findings as some sort of artefacts arising, for instance, during agony or after fixation. All the sections showed the same peculiar features, however, and all attempts to uncover the "artefacts" have so far only confirmed the observations already made. Further, a couple of observations were made that will be mentioned later on.

The measures of control required were as follows:

1. The findings had to be checked in several animals from different mothers.
2. It had to be demonstrated that the emigrated nuclear chromatin really was nuclear chromatin and not, for instance, cytoplasmic chromatin or some entirely different substance with the same staining properties as the contents of the nuclei.
3. The cytoplasmic columns observed here must not be observable elsewhere outside the sensory epithelium, and they must be present in all cristae.
4. The building up of the system should be of such a character, preferably throughout, that study of the structure alone would be sufficient to exclude that the formations here described merely signified shrunk, coagulated, constituents of the endolymph. Preferably they should be demonstrable through staining properties of the columns or network, or through other properties.

5. It would be desirable to find, in other fields of histology, features parallel to these observations.

6. It was desirable that the properties depending upon the building-up process here observed (and preferably also stages of this building-up process) be demonstrated in the cupulae of adult animals in the native state and after freezing-drying treatment.

7. It was desirable to be able to exclude, or render improbable, the possibility that the cytoplasmic and nuclear peculiarities here described might have originated during agony or after fixation.

Points 1, 3 and, in part, 4, have already been commented on confirmatively in the preceding paragraphs.

*Point 2.* The Feulgen staining is a most elective method, after which only the nuclear chromatin is stained (desoxyribonuclein). If a supposedly nuclear mass turns out Feulgen-positive it is safe to consider it a nuclear mass. In the present material, as mentioned, a great number of sections were stained by the Feulgen method and turned out to be Feulgen-positive. The results obtained with gallo-cyanin chrome-alum staining supplement in an excellent way the findings obtained by Feulgen staining and confirm experiences already gained. About every tenth section was stained with gallo-cyanin.

Under these conditions it was not necessary to resort to the measuring of ultraviolet absorption spectra to establish the findings reported.

In passing, it should be mentioned that the nuclear migration could be seen distinctly on phase contrast microscopy of unstained deparaffinized sections, and that it could be seen also in micro-incinerated sections.

*Point 3.* The cytoplasmic columns were found in all the cristae and, by a certain system, also on the utricular maculae, but never outside these structures. The columns were always seen to arise from the region between the outer cells on one side of the crista to the outer cells on the other side. The wedge-shaped cell block which delimits the sensory epithelium from the ordinary parietal epithelium showed no columns.

*Point 4.* Architecturally the columns were characterized in so many ways that it was out of the question that they might merely signify some coagulated amounts of endolymph. For one thing, the pegs were always seen within the columns, never outside, and the columns stained in every way like cytoplasm; furthermore, under the phase contrast microscope they could be made out in the un-

stained sections. For additional certainty, the author has studied the "columns" in micro-incinerated sections, which showed evidence of the same formation of columns, as here the protoplasmic elements in the lumen stand out because of their inorganic elements. This is of interest because in the cristae of adult animals the subcupular zone is rather poor in minerals, in contrast to the peripheral part of the cupula, which is somewhat richer in minerals.

*Point 5.* The developmental mechanism of the cupula here described is in principle quite in keeping with our knowledge concerning the cellular processes through which a number of other tissues develop. In numerous fetuses of various species the tissues and their anlagen have been examined at the early stages of embryogenesis, that is, before the formation of the mesenchyma, and in this way Szily,<sup>15</sup> the first, in 1908, and also Studnicka<sup>11, 12, 14</sup> besides various other authors, discovered a number of hitherto unknown phenomena, which now are considered so positively established that they are mentioned in the elementary textbooks.

In many regions, these investigators found an often extensive syncytial protoplasmic network without nuclei ("mesostroma"). Subsequently, nuclei (cells) migrate into this mesostroma, giving rise to the mesenchymal tissue consisting of anastomosing cells. Thus, for instance, Studnicka described the development of the extensive subcutaneous, non-nucleated "mesostroma" in *lophius* larva in the following well-defined phases:

- (1) Simple, long, protoplasmic, intercellular (or interepithelial) "bridges." These give rise to the formation of
- (2) an exoplasmic network, devoid of nuclei, that again turns into a compact, cell-free, gelatinous tissue, which later is
- (3) invaded by nuclei (cells).

A more detailed review of the appertaining comprehensive literature would fall outside the scope of the present work, so that for such details the reader will have to be referred to the original papers and reviews.\*

A number of details reported in the literature likewise corroborate the analogy. In keeping with this, the shark fetus appears at a late stage to present a reproduction of this developmental mechanism, which otherwise (e.g., in *Selachii*) is observed in the early phases of the embryogenesis.

---

\*Handb. d. Mikr. Anat. 1929, Vol. 2; Broman & Häggquist: *Lärobok i Histologi*, Stockholm, 1945.

In this way, then, an ectomesostroma would be formed, that is, a non-nucleated syncytial protoplasmic (exoplasmic) network of ectodermal origin that later is invaded by nuclei. Nuclear deformities as described above have not been reported in the literature, nor do the various authors appear to have paid any particular attention to the nuclear structure and its variations. As mentioned already by Szily,<sup>15</sup> an ectodermal material as the origin of the "mesostroma" is of frequent occurrence, even though mesodermal tissue as a rule constitutes the mother tissue. In ganglion cells in *Lophius piscatorius*, *Gadus* and others, Holmgren<sup>5</sup> (1899) and Hydén<sup>6</sup> have been able to demonstrate that the nuclear membrane under certain conditions opens and that parts of the nuclear chromatin mass then may be expelled through this opening. According to Hydén, the expelled chromatin serves a functional purpose in the cytoplasm.

*Point 6.* This point will be dealt with in detail in a subsequent paper.

*Point 7.* While the "snapshots" presented here presumably are to be looked upon as definitely established, the question still remains whether they reflect some *intravital processes*, or whether they (a) appeared first postmortem (agonal phenomena), or whether (b) they were due to the fixation.

(a) The fetuses were placed in the fixation fluid while still alive, with the snout cut off and the cranial roof split open. Thus access was given to the broad, free space surrounding the brain. This cavity is separated from the labyrinthine cavities by paper-thin septa of cartilage. In one case, by accident, an opening was made directly into a labyrinthine cavity, without giving rise to altered features. It is reasonable to think that the fixation has been practically instantaneous, as was evident, for instance, from the lens of the eye turning milk-white within a few seconds.

The good fixation of the other tissues (including the labyrinthine walls) with the normal nuclear pictures throughout indicates a rapid fixation. Agonal changes in one labyrinthine set of sensory epithelia would be expected to turn up also in all the other sensory epithelia, but changes of the type described above are seen only in the epithelia of the pars superior, being altogether absent in the well-fixed and unbroken sensory epithelia of the pars inferior.

Agonal changes of this character have never been described in any tissue, neither labyrinthine nor other (gelatinous) tissues. So it does not seem very likely that the nucleocytoplasmic features described previously might have been due to agonal changes.

(b) Fixation changes are well-known in histology. They may appear in the form of artificial outlines, shrinkage, etc., and their possibility is always to be kept in mind in judging histological findings, in particular when these are of the character reported here.

*Artificial structures* or the appearances of false nucleoid bodies may be ruled out rather positively on account of the uniform picture obtained with various staining methods, especially the Feulgen method, and the findings under the phase contrast microscope suggest the same.

*Shrinkage.* Pronounced shrinkage of the cupula may conceivably produce styloid cytoplasmic bodies, but it seems highly improbable that the nuclei in this way may become so much deformed as shown by the photomicrographs here. Nuclei are more apt to break into pieces than to undergo such a pronounced deformation. In would require a most violent shrinkage to produce an extension which in itself would be sufficient for such a deformity. In appraising the possibilities of shrinkage it is important to keep in mind that only the shrinkage that may have been produced by the fixation fluid is of any interest in this connection.

Further, with such a long fixation period as was employed, the nuclei may hardly undergo any particular deformation in the subsequent treatment of the specimens.

The later treatment includes, among other things, the action of alcohol upon the specimen prior to its embedding in paraffin, and this results in a quite considerable shrinkage, for instance, of the gelatinous soggy masses in the labyrinth. This is plainly evident from the photomicrographs, but, as mentioned, it may hardly affect the form of the already fixed nuclei to any extent worth mentioning.

So, it is the effect of the fixation fluid upon the specimen which is the decisive factor in the possibility of such a hypothetical feature of shrinkage in the deformation of the nuclei.

The fixation fluid consisted of equal parts of 10% formalin and 5% (pure) mercuric chloride solution.

In the concentration here employed, formalin is known to cause an increase in volume of the tissue under treatment. This has been established and estimated quantitatively by various authors (e.g., Stowell<sup>8</sup>).

Mercuric chloride was present in the fixation fluid in a total concentration of 2.5% (with an osmotic pressure lower than that of a 0.5% sodium chloride solution). The real concentration of the

substance was lower yet, as some of the mercuric chloride was reduced by the formalin into insoluble, indifferent, mercurous chloride (calomel).

In the concentration employed here and with its low degree of dissociation, mercuric chloride cannot bring about such a pronounced shrinkage of the specimens. Indeed, everything indicates that in this fixation fluid the specimen does not undergo any shrinkage worth mentioning.

Some experiments performed by the writer with a view to this possibility show that if the gelatinous labyrinthine tissue undergoes any shrinkage whatever in this fixation fluid, this is so slight that it may be observed only with difficulty (for instance, in the cupula). That such tissues under the subsequent treatment undergo pronounced shrinkage has nothing to do with our question (for variations in the volume of the tissue at various stages of the preparation and in various chemical fluids, see Stowell<sup>18</sup>). The absence of shrinkage of the tissues in this fixation fluid has been confirmed, among others, by Dr. Thydsen Meinertz, who often uses it for the fixation of fetal tissues, and this quality was also the reason why it has been employed in the present experiments in preference to other fixatives.

Additional evidence to this effect is found also in the following observations:

The shrinkage of the cupula seen in the stained section appears chiefly to be limited to the peripheral fibrillary zone of the cupula. In several specimens (Fig. 2, 3 and 6) the subcupular zone appears so broad that it hardly may be imagined to have been wider *in vivo*. This is corroborated by the study of the same zone in a frozen-dried specimen from a cod.

As a subcupular zone alone is of interest in this connection, and as it has not shrunk to any particular extent even in the finished preparation, it seems improbable for this reason too that the shrinkage of the cupula on the border of the crista might be the cause of the phenomena. For during the period in which the crista cells might have been susceptible to shrinkage they were apparently not exposed to any such influence.

This gives rise to another question, namely, why this deep zone may avoid shrinkage when the peripheral cupular zone undergoes marked shrinkage. Perhaps the answer is to be found in part in the rich mineral content (incinerating products) of the subcupular zone at this stage of life. This richness in salts thus counteracts a possible osmotic hygroscopic effect of the fixation fluid. Physiologically

Acanthias is a uremic organism with a very high blood urea level; and thus it may be that a high urea content in that part of the cupula which is supplied most abundantly by the blood stream may be of some significance in this respect. Concerning this problem, however, any statement will be merely rough guesswork. The width of this zone is clearly evident from the photomicrographs, and it is not due to oblique section of the specimen.

As to the possibility of *other fixational artefacts*, it is to be pointed out again that nowhere in any of the specimens does any of the sensory epithelia in the sacculus (macula sacculi, lagena and neglecta) show changes similar to those described in the crista even though these sensory epithelia are just as intimately connected with their voluminous gelatinous covers. If the cellular phenomena were of simple physicochemical character, depending upon the treatment of the specimens, we would expect to meet with uniform species in all the other sensory epithelia too. For the sake of completeness it is to be mentioned that in these specimens the otoliths at the most were present as scanty microscopic crystals.

Furthermore, as far as the writer knows, the preparation of histological specimens has never yet resulted in the appearance of phenomena like those described above, and thus it is not likely to have done it here.

The histological peculiarities observed here all appear to have been part of a complicated process made up of several distinct stages. Thus, for instance, the "nuclear emigration" is preceded by a "preparatory" rearrangement of the structure of the chromatin. This, together with the regular appearance of the processes within a very short period of life (within which it is reasonable to assume that the cupula has to be built up from the sensory epithelium), makes it rather probable that here we are meeting with something other than fixational artefacts.

It is further to be mentioned that by cutting the sections in a particular way allowing the subcupular zone to appear very wide (and at the same time keeping the knife from meeting the usually pronounced and sudden changes in consistency at the transition from the lumen to the firm fibrous crista) it is practicable to see similar signs of cellular emigration and formation of a similar subcupular cytoplasmic reticulum in freshly extirpated and frozen-dried ampullae from adult animals (cod) in unstained sections under the phase contrast microscope. Here the signs are far more scanty. With this form of treatment the technical artefacts are less frequent and usually of another character. On the other hand, when two methods

of treatment as widely different as these give concordant results, the validity of the findings is corroborated.

But this does not mean that the specimens described here show no distinct evidence of the thoroughgoing effect of the fixation fluids employed. Thus, besides shrinkage of the peripheral part of the cupula, the cytoplasm of some of the superficial cells has undergone shrinkage or become irregular in outline, without these changes apparently involving any definite type of cells or stage of the cellular processes. Undoubtedly these changes have occurred at a stage of treatment later than the state of fixation.

#### DISCUSSION

It may seem rather strange that such a stable structure as a labyrinthine sensory epithelium shows evidence of such thorough cellular peculiarities as appears to be the case here.

It is to be kept in mind, however, that the sensory epithelium has *two* functions, either of which is predominant during a certain period of life. In the fetus the labyrinth is built up successively, with the terminal formation of the complicated gelatinous structures that render the organ capable of function.

The cellular processes described above appear quite preponderantly to take place within the relatively short period of life when the fetus measures from 15 to 19 cm in length. In this period, therefore, the demands made on the sensory epithelial cells (being of anatomical nature) are fundamentally different from the later demand, which is chiefly physiological; and therefore the histological picture differs so much in the two periods of life.

As a matter of fact, biologically it is difficult to conceive that the entire building-up of the complicated cupula may take place merely from the cells situated at the site of the sensory epithelium. It is to be kept in mind that the formation of the fibrils in the cupula, the orientation of these fibrils, the distribution of the intercellular substance, etc., have to be brought about by forces acting on all the parts of the cupula though chiefly on the part corresponding to the surface of the sensory epithelium. In large ampullae (e.g., *Laemargus borealis*) the part of the cupula most distant from the crista is located about 4 mm from the sensory epithelium (in *acanthias* about 1.2 mm). Even on the presupposition that the cupular mass is formed as a finished structure on the surface of the sensory epithelium and then shifts peripherally, it still will be difficult to imagine that a completed structure may shift so far, especially because the zone in which it is located (just beneath the roof of the ampulla) makes its own mechanical demands upon this section.

Therefore, the writer thinks that the immigrating cells enter into the formation of a fibrillary network, in which their task is partly to produce fibrils, partly to arrange them expediently, and partly—in co-operation with the cells located in the crista—to produce and determine the amount of interfibrillary substance.

The way in which the cells emigrate is far more peculiar. In particular, it is a striking fact that during this process the nuclei apparently lose their structural properties, appearing uniformly dark and staining deeply. But this structural change need not necessarily signify a reduction in, or cessation of, their living capacity, as is evident for several reasons.

For one thing, the fixation might have affected the abnormal type of nucleus differently from the other round nuclei. Thus, for instance, a rod-shaped nucleus presents a far greater relative surface than does a spherical nucleus. Furthermore, it seems conceivable that some condition in the nucleus different from its normal state may have modified the result of the fixation as compared to the other nuclei. But this is merely speculative consideration.

In the next place, an apparent lack of structures may turn up at some decisive juncture in the life of the nucleus, especially during mitosis.

On examination of the fetal cornea in the triton and axolotl fixed in quite the same way as our specimens, the final phases of mitosis may show a scarcity of structures increasing to complete lack of structures in the daughter nuclei in their statu nascendi. This scarcity of structures may be associated with a strong or weak capacity for staining of the nuclear elements (staining with hematoxylin). The lack of structures here does not signify any avital state.

In this connection another question turns up: that of the relation of the nuclear processes to mitoses in the sensory epithelium. Mitotic figures are by no means infrequent in our specimens. A priori the possibility cannot be excluded that the emigrating chromatin masses originate from a daughter nucleus (telephase), and perhaps the lack of structure in the chromatin masses is to be looked upon from this angle.

It is to be pointed out that not all the nuclei situated in the subcupular zone are devoid of structure. Often nuclei are encountered that have lost all chromatin connection with the sensory epithelium and yet show a good structure. We may describe the nuclei in question as necrobiotic,\* not necrotic.

\*"Necrobiotic" signifying here necrosis-like appearance due to reversible processes.

At present it will be safer merely to establish that the emigrated nuclei are poor in structure and rich in chromatin, and that these peculiarities have to be taken to signify a state of the nuclei deviating from the normal one. Summarizing the above, it may be said biologically to be quite reasonable that the young cupula is built up by means of immigrated cells (or chromatin and cytoplasm), whereas it still is difficult to understand the peculiar way in which the cellular elements have preferred to emigrate. These findings constitute in themselves an unusual feature, and that means a not inconsiderable objection to their validity. Still, the writer thinks, when all the pros and cons are put on the scale, the outcome can lead only to the above conclusions.

Where similar observations have not been reported before, it may perhaps be due in part to the fact that these findings can be made only within a relatively short period of the fetal life, and this reduces considerably the chance of meeting with these phenomena. Furthermore, perhaps, it may be due to particular characters of the experimental animal. Here we are dealing with a viviparous fish, that is, a fish whose young require no capacity for labyrinthine function during the period while their cupulae are being built up. Finally, this animal shows an unusually high urea concentration in the tissues; and its skeleton is free from calcium.

The histogenesis of the cupula elucidates not only the finished structure of the cupula, but it also warrants the conclusion that the relation between the cupula and the sensory epithelium is far more intimate than previously indicated histologically.

We may even imagine the possibility that the subcupular zone ought to be referred to the sensory epithelium and considered as an integrant part of this, and that the processes we have seen taking place on the border between the sensory epithelium and this zone really ought to be interpreted as *intra*-epithelial processes. In this way they also may be understood and explained more readily. Certain findings, which will be mentioned in the following paper, lend additional support to this view.

#### SUMMARY

The author has tried to show that the ampullary cupula is formed from the sensory epithelium of the crista as follows:

From both layers of cells in the sensory epithelium long cytoplasmic columns are extended out into the lumen. These are seen to divide in the lumen and enter the formation of a syncytium-like network, corresponding to the findings in the earlier stages of embryogenesis reported by Szily and Studnicka.

After intranuclear structural changes, nuclear mass migrates out into this network. The nuclear mass emigrates in the form of peg-like chromatin bodies without structures; when it has become free of the sensory epithelium, it resumes its roundish-oval form.

In several specimens, the luminal globular chromatin bodies show chromatin structures. In this way the subcupular zone is formed, and it is encountered also in the cupulae of adult animals.

This layer gives rise to the peripheral fibrillary cupular zone.

The demonstrated intimate connection between the cupula and the sensory cells makes it possible to understand the cupular structure in adult animals and get a better idea of the mechanism in the response of the sensory epithelium to stimulation.

UPLANDSGADE 36, B.

#### REFERENCES

1. Einarson, L.: Method for Progressive Selective Staining of Nissl and Nuclear Substance in Nerve Cells, *Am. J. Pathol.* 8:295, 1932.
2. Farkas B.: Das Gehör der Fische und die cristae acusticae, *Acta Otolaryng.* 24:53, 1936.
3. Fell, H. B.: The Development in Vitro of the Isolated Otocyst of the Embryonic Fowl, *Arch. f. exper. Zellforsch.* 7:69, 1928.
4. Herzog, E.: Ueber die Entstehung der Otolithen, *Zschr. f. Hals-Nas-Ohrenheilk.* 12:413, 1925.
5. Holmgren, E.: Kurze vorläufige mitteilung ueber die Spinalganglien der Selachier u. Teleostier, *Anat. Anz.* 15:117, 1899.
6. Hydén, H.: Die Funktion der Kernkörperchen bei der Eiweissbildung in nervenzellen, *Zschr. f. Mikro-Anat. Forsch.* 54:96, 1943.
7. Kolmer, W.: Ueber das Verhalten der Deckmembranen zum Sinnesepithel, *Arch. f. Ohrenheilk.* 116:10, 1926.
8. Stowell, R. E.: Feulgen Reaction for Thymonucleic Acid, *Stain Technol.* 20:45, 1945.
9. Stricht, N.v.d.: L'istogenese des parties constituants du neuroepithelium acoustique, *Arch. de Biol.* 23:541, 1908.
10. Stricht, N.v.d.: Les membrânes tectrices des crêtes et des tâches acoustiques, *Arch. de Biol.* 31:299, 1921.
11. Studnicka, F. K.: Ueber einige Grundsubstanzgewebe, *Anat. Anz.* 31:497, 1907.
12. Studnicka, F. K.: Das Mesenchym und das Mesostroma der Froschlarchen u. deren Produkte, *Anat. Anz.* 40:33, 1912.
13. Studnicka, F. K.: Die Otoconien, Otolithen u. Cupulae terminales im Gehörorgan von amnocytes und petromyzon, *Anat. Anz.* 42:529, 1912.
14. Studnicka, F. K.: Noch einmal die Cytodesmen des Mesostroma und die Grundsubstanz, *Ztschr. f. Zellforsch.* 4:365, 1926.
15. Szily, A.: Ueber das Entstehen eines fibrilaren Stützgewebes im Embryo u. dessen Verhältnis zur Glaskörperfrage, *Anat. Hefte.* 35 Heft. 107:649, 1908.
16. Werner, Cl. F.: Die Cupula im Labyrinth der Fische, *Ztsch. f. Zellforsch.* 4:459, 1926.
17. Werner, Cl. F.: Das Labyrinth, Leipzig, 1940.
18. Wittmaack, K.: Ueber d. Tonus Sinnesendstellen des Innenohres, *Arch. Ohr-Nas-Kehlkopfheilk.* 114:278, 1926.
19. Wittmaack, K.: Nachtrag zu meiner Arbeit ueber Aufbau u. Funktion d. Cupula, *Acta Otolaryng.* 24:424, 1937.

### III

## STUDIES ON THE COMPLETED STRUCTURE AND MECHANISM OF THE CUPULA

THURE VILSTRUP

COPENHAGEN, DENMARK

In continuation of the studies reported in a preceding paper,<sup>13</sup> the author will present here the results of his studies on the structure of the cupula in the adult animal, together with a few supplementary and controlling experiments performed on living animals.

The cupula, as it has been mentioned, is a gelatinous, approximately ovoid, formation, one end of which is shaped as an impression of the free margin of the crista to which it is attached. It extends throughout the ampulla, reaching thus from the crista to the roof of the ampulla.

According to our present knowledge of the macroscopic anatomy, histology and function of the cupula, it is reasonable to assume that the cupula is a mobile formation for the purpose of acting as a mechanical intermediary in the exchange of energy from the movements of the endolymph to the discharges of the central cells in the efferent nerves.

Angular, accelerated movements of the head are associated with a spatial shift of the membranous semicircular canal, and the tendency of the endolymph to stand still causes a shift in the relation of its mass to the walls of the semicircular canals. As the cupula is mobile, floating freely in the lumen of the ampulla and fastened only to the crista, it will follow such a current in the endolymph and thus become deflected. This deflection affects the sensory epithelium so as to give rise to impulses that may be transmitted to the efferent nerve. Here they may be measured and registered,<sup>8</sup> and we know that these impulses in the central organ are transformed in such a way that peripheral functional muscular contractions, the so-called ampullary reflexes, are elicited. These are characterized, among other things, by their rapidity; and it has been demonstrated that the reflex arches presumably comprise but two synapses.<sup>3, 10</sup>

---

From the Institute of Histology at the University of Lund, Sweden, and the Department of Oto-Rhino-Laryngology at the University Clinic of Lund, Sweden.

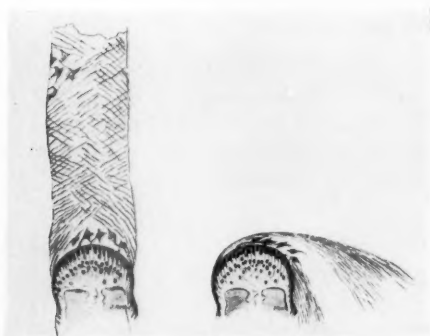


Fig. 1.—Schematic drawing showing the structure of the erect and deflected cupula in a frozen-dried cod ampulla.

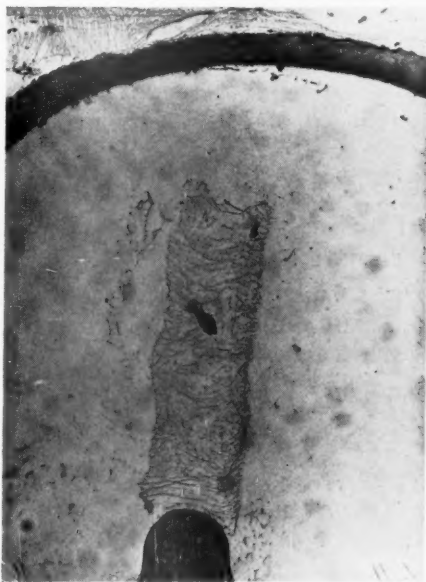


Fig. 2.—Photomicrograph of section through frozen-dried cod ampulla. (Toluidine blue stain;  $\times 100$ .)

The investigators who primarily have led us to this conception are Steinhausen<sup>12</sup> and Dohlman.<sup>4</sup>

Steinhausen injected India ink into the semicircular canals in living and recently killed pikes and obtained in this way the negative picture of the cupula. Thus he established the actual size of the cupula and its mobility. He further found a certain rough agreement between the immediate position of the cupula and the muscular reflexes.

Later, Dohlman repeated the Steinhausen experiments with improved technique and extended them by injecting India ink into the cupula through the crista itself. After this it had to be taken as proved that the cupula extends to the roof of the ampulla and that its movements proceed like a "swing-door in its frame" (Steinhausen).

The writer has performed a number of experiments using the Steinhausen-Dohlman technique on living sharks and likewise found a very close concordance between the position and movements of the cupula and the muscular reflexes of the animals. In a subsequent paper it will be mentioned that the writer also has been able to show that the "reflex reversal" probably occurs at the very moment when the cupula passes its 0 position and that a given position elicited by an ampullary reflex is preserved exactly as long as the cupular flexion is maintained. It is further pointed out that there is a certain conformity between the degree of the cupular flexion and the intensity of the elicited reflexes.

*The building-up of the cupula and its connection with the sensory epithelium* are still somewhat obscure. Originally the cupula was looked upon as a small, firm and immobile structure, riding on the crista (e.g., Retzius,<sup>17</sup> 1884). Its connection with the sensory epithelium is generally taken to be established by means of long "cilia," projecting from the so-called hair-cells of the sensory epithelium into the gelatinous mass of the cupula, thus being deflected with this. Many suggestions have been made concerning the nature of the "cilia," without any agreement being reached. Here it is to be mentioned that Wittmaack<sup>18</sup> says that the cilia often are split irregularly, with "protoplasmakügelchen" attached, and that their structure perhaps is not as simple as previously assumed.

It is the prevailing view that each "cilium" in the cupula is surrounded by a homogeneous gelatinous mass (e.g., Kolmer,<sup>7</sup> 1913; Retzius, 1884), and several authors (among others, Wittmaack,<sup>18</sup> 1926, who in several respects appears to have advanced farthest in the studies on the more delicate structures of this organ) claim that the fibrils observed in the cupula are continuations of sensory hairs



Fig. 3.—Photomicrograph of section through frozen-dried cod ampulla. (Toluidine blue stain;  $\times 45$ .)



Fig. 4.—Photomicrograph of section through frozen-dried cod ampulla showing sensory epithelium and subcupular zone. In the middle and to the left emigrating cells are seen. (Toluidine blue stain;  $\times 1000$ .)

("cilia"). Werner<sup>14</sup> (1940) is rather inclined to subscribe to this view.

As all the cupulae hitherto described were seen in specimens which beforehand had been treated with chemical fixatives—the effect of which on the structure and size of the various elements is radical and rather obscure as to details—the resulting pictures need not correspond to the actual appearance of the cupula. Thus the cupulae observed in adult animals have been shrunk markedly, and in most specimens they have been nearly gone. This led on to, for instance, Bowen<sup>2</sup> taking the cupula to be an artefact and assuming the presence of kinocilia instead of sterneocilia in a mobile frame. In all successful sections of chemically fixed cupulae, this structure is seen to be built up of parallel lighter and darker stripes, and the dark lines are generally taken to represent fibrils, the light lines interfibrillary substance. In these preparations the fibrils always radiate from the sensory epithelium towards the periphery of the cupula.

As to the interpretation of the microscopic features of the adult cupula, especially its connection with the sensory epithelium, the theories advanced so far are uncertain and conflicting. Still, the greater majority of investigators appear to agree that it seems most reasonable to assume that the so-called "cilia," originating from the hair-cells, continue directly in the cupular radiate fibrils. Thus a deflection of the cupula will bring about a deflection of the cilia, conveying in this way an impulse to sensory cells. Hitherto, however, it has not been practicable to study these cilia in detail. Still, as has been mentioned, Wittmaack<sup>14</sup> and others found that the cilia often appear to be split in their middle and peripheral parts, and that they often presented an irregular outline, being connected with small protoplasmic globules or lumps that were taken to be artefacts. These protoplasmic lumps appear not to have been associated with the uncertain signs of the presence of cellular nuclei and protoplasm which some investigators after Retzius think they have seen in the loosely built-up subcapsular space between the sensory cells and the fibrillary part of the cupula. Most investigators have considered the presence of such formations as so improbable that they have refuted these findings as due to artefacts. Presumably these "artefacts" have looked very dim and irregular, as they are apt to unless particular care is taken to preserve this very zone from injury.

Further investigation into the structural aspects of the cupula is desirable for the following reasons:

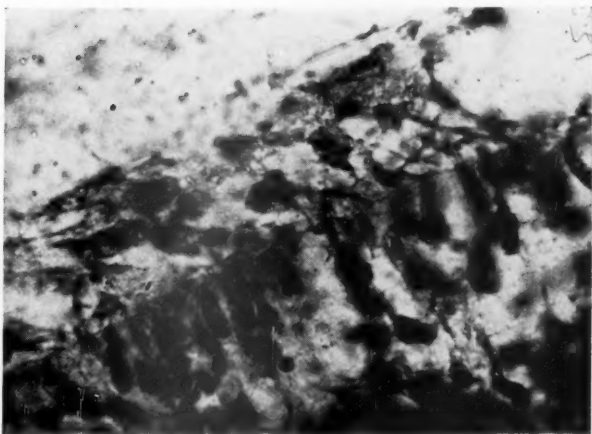


Fig. 5.—Photomicrograph of section through frozen-dried cod ampulla showing the sensory epithelium and subcupular zone. Note the subcupular reticulum and its connection with the sensory epithelium. (Toluidine blue stain;  $\times 1000$ .)

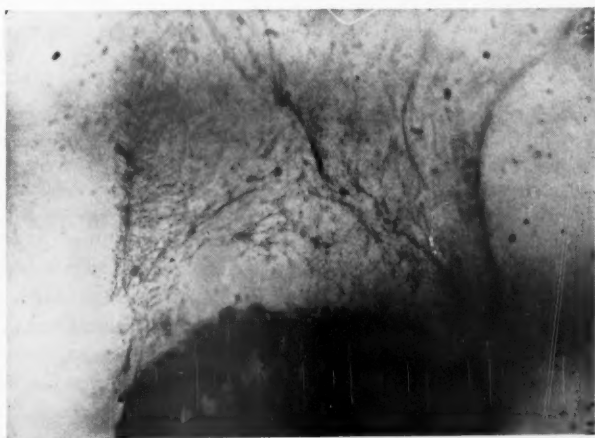


Fig. 6.—Photomicrograph of section through frozen-dried cod ampulla showing sensory epithelium and basal section of the cupula. In order to make the latter more distinct the picture was somewhat over-developed. The fan-shaped marginal fibril bundles in the cupula are more distinct to the right. (Toluidine blue stain;  $\times 325$ .)

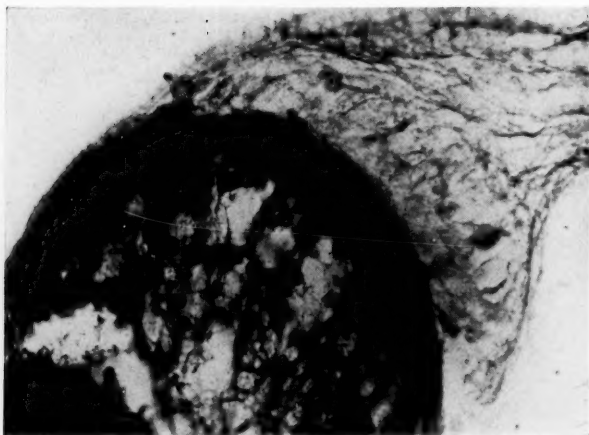


Fig. 7.—Photomicrograph of section through frozen-dried cod ampulla showing sensory epithelium and basal part of the slightly deflected ampulla. Note the water-clear, elastically stretched marginal fibers at the left. (Toluidine blue stain;  $\times 425$ .)

Since the classical studies on the labyrinth were reported, histology has been advancing rapidly, and today new methods are available that in many fields offer considerable advantages over the previous ones.

Thus a number of problems, including histophysical ones, may be explained but very inadequately by means of the previous conception of the cupular structure.

One of these problems concerns the sensory cell and its change in form under deflection of the cupula. If the cilium of a sensory cell continues far out into the cupula, perhaps directly as a cupular fibril, it will be deflected with the cupula, and at the same time all the sensory hairs on one side of the crista will be stretched. If these sensory hairs are not elastic—and nobody has yet suggested their being elastic—this stretching will be transmitted to the sensitive sensory cell, which in this way will undergo deformation. This deformation may appear in the form of stretching in the direction of the traction, the thick goblet-shaped sensory cell thus becoming elongated and slender. The sensory cell may be estimated under complete cupular deflection to be stretched to about twice its normal length—still provided that the cilia are not elastic. A priori, such a stretching may be refuted as nonphysiological and most improbable.

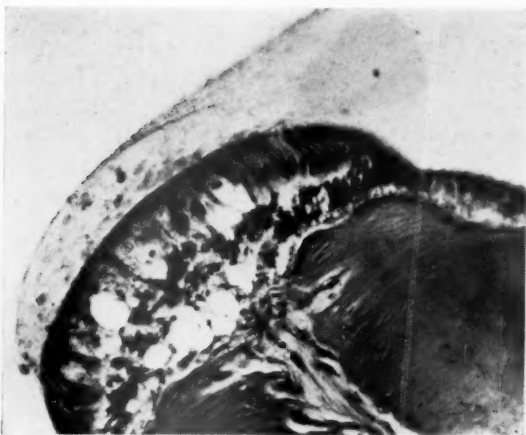


Fig. 8.—Photomicrograph of section through frozen-dried cod ampulla showing the cupula entirely deflected, without altering the form of the subcupular zone essentially. (Toluidine blue stain;  $\times 1000$ .) (Same section as shown in Fig. 9.)

As hitherto the nature of the cilium has not been recognized clearly, and it has rather been taken for granted that the cilium here is a structure analogous to other stereocilia (in which no elasticity was ever demonstrated) there was no reason whatever to take these particular cilia to be elastic. So, simply for this reason it is essential to study in detail the phenomena associated with the deflection of the cupula.

Another feature that does not fit into the present picture is evident from the following:

Movements of the cupula do not take place alone, as was assumed hitherto, by deflection around the crista as center of the motion and as the fixed point, but also, and in a pronounced degree, by a "sliding" of the cupula over the surface of the crista or sensory epithelium. Undoubtedly this sliding takes place on the border between the two zones of the cupula. This sliding, which was pointed out by Dohlman in collaboration with the writer, may be demonstrated as follows: On intracupular injection of India ink through the crista in the way described by Dohlman, a narrow band of the cupula is stained, extending from the crista to the roof of the ampulla. If such a cupula is deflected by artificial endolymph currents, and the stained band is studied closely during the deflection, it can be seen in every



Fig. 9.—Same section as Fig. 8. Note the water-clear, elastically stretched marginal fibrils at the left. (Oil immersion.)

instance that the *cupula* shifts tangentially over the surface of the sensory epithelium. At the same time the cupula gives way and is deflected in the "classical way." The writer noticed this phenomenon first in experiments on living sharks, and since he has observed it again and again, always with the same result. The same phenomenon can be seen in the cod. The cod employed by the writer were alive until immediately before the operation, and hence postmortem changes may be excluded here as well as in the shark.

Nor can this phenomenon be due to some strong nonphysiologic agency, as the endolymph currents were produced with a glass capillary tube filled with India ink, and the cupula movements thus produced were quite slow. In 1938, Dohlman took a film showing movements of the cupula after treatment in different ways. The cupula was rendered noticeable by various contrast injections. A closer study of this film shows that the phenomenon here described can be made out in all the six cod ampullae used for the film. Dohlman stated that he had not attached particular significance to the tangential motion, which he indeed had noticed himself, as at that time he thought it might be due to some postmortem agencies.

From this film the writer has borrowed some suitable photos, shown in Fig. 15. It will be noticed that the staining with India ink does not quite extend to the surface of the crista, and that the staining commences about at the border between the cupula and the subcupular space.

The hitherto prevailing histophysical view of the cupula is not sufficient to explain the sliding phenomenon of the cupula. This phenomenon will be mentioned again in the discussion of structural changes in the cupula under its deflection.

#### PRESENT INVESTIGATION

In the rest of the author's studies more recent histological methods of examination are employed whenever possible, in particular, more modern fixation methods; methods that were not available to previous investigators in this field.

The most important of these adjuvants is the freezing-drying fixation technique devised by Altmann-Gersh. Since its first publication by Gersh<sup>5</sup> in 1932, this method has become classical in all large histological laboratories and hence it needs no detailed description here.

Numerous control examinations have been carried out to find the limitation of the method and to make sure that the findings in frozen-dried specimens actually correspond to what may be expected

to be present in life, and that this treatment does not bring about any artefacts that may not be accounted for.

Briefly, this method is based on the following principle:

The tissue is frozen instantaneously at a temperature of  $-180^{\circ}\text{C}$ . Then it is placed in a vacuum tube at  $-60^{\circ}\text{C}$ , connected with a desiccator, and here it remains until it is dehydrated. Then the tissue is directly embedded in paraffin—if necessary under vacuum—and cut in the usual way in sections of the thickness required (7-10 and  $15\text{ }\mu$ ). These sections may be submitted to micro-incineration without further treatment. Other sections are deparaffinized in xylol or toluol, after which they may be studied directly under the phase contrast microscope. These sections, however, as well as those to be mentioned further on, are most often (but not always) denatured in alcohol before being submitted to further treatment. The denatured sections (which under the phase contrast microscope look quite like the nondenatured sections) may be stained in the usual way.

At the initial low temperature the tissue is frozen instantaneously, and the resulting ice crystals are submicroscopic, and therefore any possible tears in the tissue produced by these crystals likewise are submicroscopic. Owing to the dehydration, the substances employed afterwards can have no influence upon the water phase of the specimen, and thus shrinkage is excluded. The low temperature at which the dehydration takes place prevents any shifts of the substances in the specimens. Thus the finished specimen may be said to contain the stroma of the original tissue in unchanged form.

In this connection it is of particular interest to know whether this treatment tends to precipitate stroma-like configurations in specimens in which they are not encountered beforehand.<sup>9</sup> Concerning this point, Glimstedt<sup>15</sup> stated: "In properly fixed specimens treated after this method, the ground cytoplasm shows no microscopically noticeable structure." According to him, this applies, for instance, to the tissue of the vitreous body of the eye, and, according to Sjöstrand,<sup>11</sup> to parts of the kidney tissue.

In principle this method was devised by Altmann,<sup>1</sup> and since it has been further elaborated and improved by various investigators.<sup>5, 6, 16</sup> A drawback of this method, as in other methods, is that the lipoids in the tissue to some extent are dissolved and removed with the removal of the paraffin, but this may hardly play any particular role in the present work.

A brief survey of the Altmann-Gersh freeze-drying technique is given by Sjöstrand,<sup>11</sup> to which the reader is referred for



Fig. 10.—Photomicrograph of section through the crista and cupula from adult shark, chemical fixation, showing subcupular zone empty except for a few fine threads. (Toluidine blue stain;  $\times 150$ .)



Fig. 11.—High magnification of the area to the left in Fig. 10. (Oil immersion;  $\times 1750$ .)

details of the technique and to the requirements which the specimens have to meet before their freezing. The writer has followed the principles given by Sjöstrand. Here it is merely to be emphasized that in the present work all the specimens treated in this way have been very small, consisting of ampullae removed from cod that were killed immediately before the freezing.

Efforts have been made to cut down the interval between the death of the animal and the moment of the freezing of the specimen to the least possible, and it was found practicable to limit this interval to about five minutes, which is about as long as permissible. For a sort of control, the ampullae were removed from two cod still alive, reducing the interval to about 15 seconds. The findings in these two ampullae differed in no way from those in the other ampullae.

The deep freezing was performed by means of isopentane, chilled beforehand in liquid nitrogen, and the dehydration period amounted to three weeks, in a few cases four weeks. At the embedding in paraffin the ampullae were placed carefully so that the sections as far as possible were made perpendicular to the longitudinal axis of the crista. This orientation of the specimen, which was performed under a magnifying glass, was facilitated by removing the ampulla together with as long a piece of the membranous semicircular canal as possible. The ampullae thus removed consisted exclusively in anterior and lateral ampullae. In the cod the posterior ampulla is located so deeply that its removal takes far too long. Furthermore, this ampulla may hardly be removed without some mechanical injury, and hence it was not employed at all. In the staining of sections aimed especially to show the fibrillary and cytoplasmic structures the best results were obtained with toluidine blue stain (pH 5.5), while the sensory epithelium proper and the nuclear elements in the subcupular space were stained with hematoxylin-eosin, gallocyenin or by the Feulgen method.

It is to be mentioned that for the sake of additional certainty, the writer has controlled his results in studies on the fibrillary structure of the cupula in another way, too: Fresh ampullae were extirpated and laid open, after being ligated immediately at each ampullary ostium. Then the cupula was spread on a slide by means of a fine brush, with the perilymph and endolymph adherent to this structure (one drop). A cover slip was placed on top of the preparation, which was then examined directly under the phase contrast microscope. In this way it was practicable to study the truly native cupula, and after staining with toluidine blue the fibrils observed already in the native cupula were seen again. The picture thus ob-

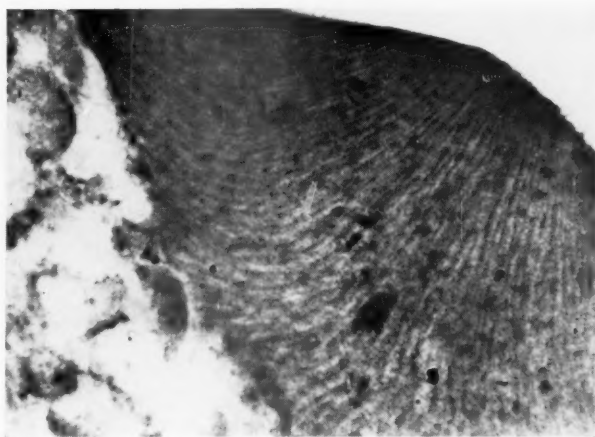


Fig. 12.—Photomicrograph of section through the cupula from a late shark fetus, fixed chemically. (Hematoxylin-carmin stain;  $\times 600$ .)



Fig. 13.—Photomicrograph of area in the lower left corner of the cupula shown in Fig. 10. Note the spiral twisting of the fibrils. (Oil immersion;  $\times 1750$ .)

tained of the cupular fibrils corresponded to the findings in the sections of the frozen-dried specimens, stained as well as unstained.

*Findings.* The construction of the cod ampulla on the whole corresponds to the construction of the other ampullae described in the literature. A single special feature is found at the canalicular ostium of the anterior ampulla in the form of a voluminous gland-like structure,\* which fills in part the ostium between the ampullary cavity and the recessus utriculi. This structure will not be further described here.

The free crest of the crista is covered by the sensory epithelium which does not quite correspond to the crista epithelium previously described in the shark. In the cod the sensory cells are very long, close-pack, and provided with small, dark, slightly oval nuclei, poor in structure, situated basally. In contrast to the findings in the shark, here the distribution of the sensory epithelium on the crista appears symmetrical.† The crista is covered by the cupula, the structure of which will be dealt with in the following paragraphs.

The structure of the cupula as it will be described was found in all the specimens in which the cupula was examined in the same phase of deflection, regardless of staining, etc. Thus the same structure was found in the unstained deparaffinized sections under the phase contrast microscope and even in the untreated micro-incinerated paraffin section, although less distinct here. In every instance the cupula appeared to consist of two parts: a zone adjacent to the crista, and a peripheral zone. The former is chiefly of cytoplasmic character, the latter rather fibrillary. In the following the former will be mentioned as the subcupular space (zone), the latter as the fibrillary zone.

The *subcupular zone* (Fig. 1, 7, 8 and 9) extends from the outermost sensory cell on one side of the crista to the outermost sensory cell on the other side. On the sides it is delimited by the elastic marginal fibers of the fibrillary cupula, and it is defined against the fibrillary cupula by fan-shaped bundles of fibrils arising from the margins of the sensory epithelium, converging and meeting over the middle of the crista (Fig. 1). The height of the zone is about one-sixth to one-eighth of the total height of the cupula, that is, of the distance from the crista to the roof of the ampulla. The subcupular zone extends along the crista in its entire length from planum semilunatum to planum semilunatum. How it is delimited from the

---

\*This is now being studied more thoroughly by the writer.

†See "Studies on the Construction and Function of the Semicircular Canal System" by the author (in press).

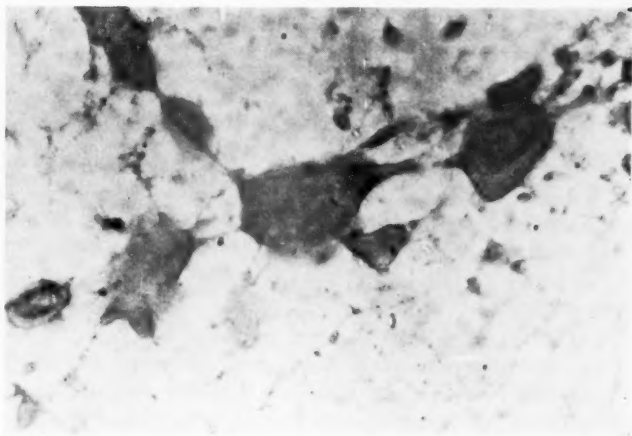


Fig. 14.—Photomicrograph of section through frozen-dried cod ampulla showing peripheral section of the cupula with stellate cytoplasmic structures anastomosing mutually and with the cupular fibrils. Round-oval shadows are seen in two of the formations (nuclear remnants?). (Toluidine blue stain; oil immersion;  $\times 1750$ .)

*plana semilunata* is still unknown (also whether it really is delimited here).

The subcupular zone is filled with a gelatinous mass that shows a distinct structure. This mass is characterized by the presence of a good deal of cell-like structures as well as fibrillary elements by means of which the observed cells appear to be "anchored" to the surface of the sensory epithelial cells. In analogy with the findings described by the writer in a preceding paper<sup>13</sup> dealing with the histogenesis of the cupula it seems reasonable to assume that these thin thread-like structures represent cytoplasmic fibers from the crista cells or cytoplasmic remnants from immigrated cells. In either instance, these threads signify the fundamental unity of the cells in this zone and the sensory epithelium. The nuclei of the cells in the subcupular zone appear not all to be structureless. With the stains employed, the cytoplasm seems homogeneous, and even though its outline often is rather indistinct, many things suggest that these cells are stellate (Fig. 4, 5 and 9) and form a reticular network which on one side is intimately connected with the fibrillary extensions of the sensory cells, on the other side with the fibrils in the peripheral fibrous part of the cupula. The transitions to either side may be followed in numerous sections with a reasonable degree of

certainty.\* The extent and form of the subcupular zone appears rather constant, regardless of the position of the cupula, as will be pointed out later on.

The *peripheral fibrillary zone* is made up of fine fibrils which under polarized light are found to be double-refractive.

*The Cupula in Zero-position.* In sections cut with the cupula in this position, that is, on cross-section of the crista, a great part of the fibrils appear to radiate from the peripheral margin of the sensory epithelium in broad fan-shaped bundles (Fig. 1 and 6). The lateral† fibrils in such a bundle enter into the lateral border of the basal part of the cupula and point axially northward. The axial parts of the two fan-shaped bundles meet over the middle of the crista, forming the borderline between the subcupular zone and the fibrillary zone. These fibrils continue gradually in the rest of the cupular fibrils which appear mostly to run parallel with these last-mentioned axial fibrils. Thus the main part of the fibrils in this part of the cupula run neither radiately from the sensory epithelium (as is otherwise seen in cupulae after chemical fixation) nor parallel with the sensory epithelium, but rather midway between these two directions. This, no doubt, is of great mechanical importance as it facilitates a change in the orientation of the entire fibrillary network, as, for instance, in deflection of the cupula (Fig. 9).

In the first-mentioned fan-shaped bundle the fibrils are very long, whereas the peripheral fibrils (situated northward) are shorter and apparently separate from each other.

No structure is seen between the fibrils.

In this section of the cupula also a few cells are seen, usually with pale "washed-out" nuclei. These cells are markedly stellate and in their many corners provided with long extensions which either anastomose with similar extensions of neighboring cells or continue directly into cupular fibrils of the usual type (Fig. 14).

In these "cells" as a rule no nucleus is seen. Now and then, however, it is possible by Feulgen staining to make out some structures maybe containing nucleoproteins. It is interesting to see that the cells observed in the center of the fibrillary zone are better preserved than those seen along the margins; and this applies especially

---

\*The frequency of cells or nuclei in the subcupular zone is much lower here than in the shark fetus.

†The terms of direction here employed are: axially: in towards the symmetrical plan of the crista; laterally: away from the symmetrical plan of the crista; northward: up towards the roof of the ampulla; southward: towards the sensory epithelium.

to the "nuclei." Presumably the cause of this is to be looked for in the histogenesis, as it seems reasonable to think that these cells have taken part in the building-up of the fibrillary cupula and, having accomplished their task, fade away in the periphery. The nature of these structures, however, has not yet been fully established.

The most northerly part of the cupula has proved so fragile in all the specimens that in the sections it is fragmentary or altogether absent. Structurally it does not appear to differ essentially from that part of the cupula from which it has been detached. In most sections the lateral delimitation of the cupula is as sharp as might be expected. As a rule, there are no marginal fibrils following the edges of the cupula in its peripheral ("northern") two-thirds. The "southern" third, as mentioned above, is provided with marginal fibrils in the form of the "lateral" fan-shaped bundles. This was the picture encountered in all the cupulae examined—a total of about 90—even though suggestions of "north-south" border fibrils were seen occasionally.

*The Deflected Cupula.* An entirely different structure with rearrangement of the fibrils is seen in the deflected cupula (Fig. 1, 8 and 9). In its main features the deflected cupula is characterized by the following: The angular bundle on the side opposite the direction of the deflection becomes more compact, and the fibrils in its apical section are extended up to several times their original length. As a consequence of this stretching the formerly deeply staining fibrils now become pale or colorless. The point from which the fibrils commence diverging is shifted considerably; in a maximally deflected cupula this point is located almost at the axial line or even a little farther towards the deflected side.

In lesser deflections the point lies nearer the origin of the fibrils. From this point the fibrils diverge in the usual way although the angle formed between the outer fibrils now is somewhat smaller than before the deflection.

The opposite V-formed fiber bundle is bent forcibly down towards the floor of the ampulla, so that any trace of the original direction is lost in the completely deflected cupula. At the same time, the fibrils spread, so that the angle between the outer fibrils is increased.

The rest of the cupular fibrils, which, as mentioned before, were found to be arranged after a trellis-like system, are rearranged and now run mostly parallel, apparently radiating from the borderline between the two cupular zones. At the same time the most southern fibrils move downwards and fill the space between the two V-formed

bundles. Because one of the fans is not unfolded completely, a "vacant space" arises that is filled with these fibrils. This evidences the expedient architecture in the above arrangement of the peripheral fibrils in the erect cupula.

Under deflection of the cupula (and probably under its shrinkage, too) the fibrils in the peripheral zone of the cupula are being rearranged, each fiber "turning," so that instead of running chiefly parallelly with the free margin of the sensory epithelium they now take a course preponderantly perpendicular to this margin.

Presumably the most important consequences of the cupular deflection is that the subcupular zone "opens" on one side (right side of Fig. 8), and that its contents are pulled gently towards the side where the deflection takes place.

From Fig. 8 and 9 it is evident that the entire contents of the subcupular zone become slightly dislocated at the deflection of the cupula, shifting parallelly with the sensory epithelium. This shift is only slight, yet sufficient to make all the fibrillary components appear deflected.

At the same time the violent stretching of the marginal fibrils exerts a pressure on one-half of the subcupular zone. How big a part of the zone is exposed to this pressure will vary with the degree of the deflection of the cupula and thus with the intensity of the impact of the endolymph against the lateral aspects of the cupula.

Thus we arrive at the explanation of the sliding of the cupula under its simultaneous deflection, as shown in Fig. 15.

Correlation of Fig. 15 with Fig. 1 affords a basis to understand how a column of India ink deposited in the space between the two fan-shaped bundles will shift under the cupular deflection.

Keeping in mind that the living cupula contains a gelatinous amorphous mass between its fibrils, it is easy to understand that under deflection of the cupula the axial part of this mass must be shifted over the border between the two cupular zones at the same time as its components are rearranged completely. The friction that has to be overcome in this process is surprisingly slight, as has been demonstrated by Dohlman.<sup>4</sup> From the writer's own experiments, however, it appears as if this friction is so marked that the inertia conveyed to the cupula under quick angle-accelerated motions is hardly sufficient by itself to overcome it.

The fact that under the impact of the endolymph the cupula slides and is deflected at the same time opens new prospects for the

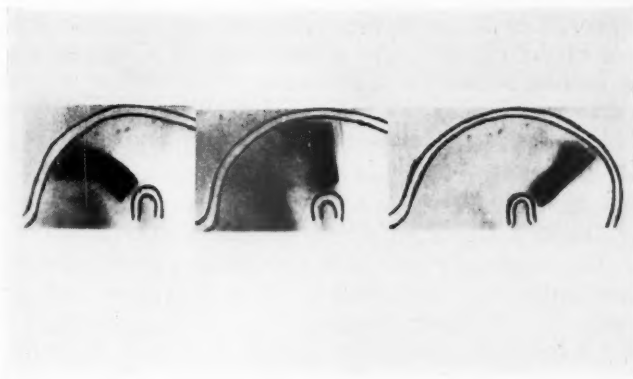


Fig. 15.—Three pictures from the same section of Dohlman's film. The outline of the ampullary wall is drawn with India ink. The cupula was injected with India ink by Dohlman's method. Three phases of the same motional deflection of the cupula are shown. Note the unstained subcupular zone; the cupula appears to be sliding rather than being deflected.

study of the cupular function. The sliding is of such a magnitude as to be clearly visible; furthermore, it exceeds by far the limit of error associated with the reading of the cupular movements. The extent of this sliding is illustrated by Fig. 15.

An additional feature is to be mentioned here. It is the writer's impression that the cupula actually is "a little higher" (in the north-south direction) than allowed by the dimensions of the cupullary roof. Correspondingly, the cupula should be "stretched" by such a high degree of flexion that the greater distance between the crista and the lateral part of the roof of the ampulla might be utilized. At the present stage, this view is very far from being proved, but it may perhaps be appropriate here to give a few reasons in favor of this view.

1. Optimal sections, showing a deflected cupula, convey a distinct impression of the distance between the outermost part of the cupula and its point of attachment being greater than observed for a cupula in the erect position. Exact measurement of this distance is rendered difficult by the circumstance that the periphery of the deflected cupulae as a rule is not faultless, being mostly frayed. The same thing is seen in micro-incinerated sections. Here the fibrillary zone is characterized by its contents of close-packed small crystals of inorganic tissue that make the outlines of the cupula stand out sharply. On studying such sections, one also gets the impression that

the above mentioned "measure of length" of the deflected cupula exceeds the direct distance between the crista and the nearest point on the roof of the ampulla. The writer's material of successfully deflected cupulae is not yet sufficient to allow decisive conclusions to be drawn concerning this point, and the wax-plate constructions required to decide this matter were not performed (see Fig. 15, showing an injected cupula in three positions).

2. Another observation made by the writer is that every fibril in the fibrillary cupula really may be considerably longer than it looks. On examination of a slide preparation of the native cupula (as described above), each fibril is seen to be twisted and slightly shortened. If the cupula is exposed even to slight shrinkage, this twisting is increased considerably and the fibril takes the shape of a spiral spring. This may be seen in cupulae after chemical fixation, where the shrinkage is by far most pronounced in the general direction of the fibrils (Fig. 10 and 13), and also in cupulae that have not stayed long enough in the dehydration tube. Furthermore, the spiral twisting of the fibrils in the native preparation increases with increasing drying of the specimen under the cover slip.

This in connection with the trellis-like arrangement of the fibrils in the cupula may conceivably indicate the condition suggested by the writer.

3. From the experiments reported by Steinhäuser<sup>12</sup> and by Dohlman<sup>4</sup> it is evident that the cupula is in close, "water-proof" contact with the roof of the ampulla, and that the India ink is able to pass the cupula as a delicate film only when the cupula is flexed almost to the maximum. This intimate connection of a gelatinous lump like the cupula is explained most readily if we assume that the gelatinous lump is "just a little too big" for its space and that it possesses elastic qualities that enable it to adjust its form after the given dimensions of this space. The "given dimensions of this space" refer to the fact that the roof of the ampulla is not altogether a section of a circle in outline. On drawing a circle with its center in the sensory epithelium of the crista and its radius equal to the perpendicular distance to the roof of the ampulla, only the axial part of the roof will be congruous with this circular arch (Fig. 15). The lateral parts, that is, those parts that still may get in contact with a strongly flexed cupula, fall a little outside this circle. The writer has measured these dimensions on projected sections from shark and cod ampullae and found the same proportions, though with small quantitative variations. No definite conclusion may be drawn from these findings, however, because the roof may be readily exposed to slight shifts or changes in form in the sectioning of the specimens.

Thus a possible "stretching" of the cupula under its deflection is not incompatible with the structure of the cupula, nor with the general construction of the ampulla. Still, the present material seems far from sufficient to allow any definite conclusions to be made in this respect.

In comparing the observations described here with the previous observations on the histogenesis of the cupula,<sup>13</sup> we meet with a striking conformity that allows further conclusions to be drawn with regard to the significance and character of the structures of the subcupular zone in particular.

Here it will be appropriate to remember the finding that in the shark fetus the crista cells project broad, protoplasmic columns out into the lumen, with preservation of the protoplasmic connection with the crista cells. Both layers of crista cells take part in the formation of this luminal product, which appears as a cytoplasmic network intimately connected with the fibrillary cupula as well as with the sensory cells.

Considering the findings in the cristae of the adult cod, we meet here with cells that are about to emigrate from the crista, and also the cells that already have emigrated and are now lying free in the lumen. The latter often seem to be connected with the crista epithelium by a long, thin thread which, in analogy to the above, has to be interpreted as a remnant of a broader cytoplasmic bridge that originally connected the emigrated cell with the crista epithelium.

Consequently, then, these "threads" may be interpreted as remnants of the originally broad cytoplasmic column through which the nuclear mass emigrated from the nuclei of the sensory epithelium (or possibly as remnants of the cytoplasmic column connecting the crista epithelium with cells that had emigrated in toto). Figure 4 shows plainly such a cell that appears to have emigrated in toto but not yet broken off its cytoplasmic mooring.

Of all this, only a minor part is seen in a chemically fixed, shrunken cupula from an adult shark (Fig. 10). The findings here may be described briefly as follows:

The subcupular zone appears empty or nearly empty. A few delicate, often irregular, threadlike structures are seen to arise from the sensory epithelium. But, as a rule, it is not possible to decide from which cells in the sensory epithelium these threads originate. Most often they end freely in the lumen, but occasionally, in suitable specimens, they may be seen to continue into the fibrillary cupula.

Cells or Feulgen-positive nuclear structures are practically never seen in this zone.

The peripheral fibrillary cupula is characterized by shrinkage and rearrangement of the fibrils. The fan-shaped bundles arising from the margins of the sensory epithelium now appear compressed like a closed fan. The axial fibers have remained in their place, aggregating in thicker bundles so that the outlines of the cupula stand out sharply. This applies in particular to the delimitation from the subcupular zone, corresponding to a more pronounced shrinkage on the "surface" (including the walls of the subcupular zone).

The other fibers are arranged in the north-south direction and are pulled down towards the border between the two zones (as seen also in frozen-dried deflected cupulae). The shrinkage has taken place chiefly in the north-south direction, and the reason for this is easy to see on employment of higher magnification or polarized light: all the fibers running north-south are shortened considerably, being twisted spirally. Owing to the other injurious effects from such treatment of the cupula, the course of the fibrils is somewhat irregular and variable (Fig. 13). On account of the thick, deeply staining bundles of fibrils in the basal part of the fibrillary cupula the possible continuation of the "fine" cilia cannot be followed through these bundles.

It was only reasonable and obvious at the finding of such thread-like formations arising from the sensory epithelium to designate them as "cilia" or "hairs" and to designate the cells from which they apparently arise as "hair cells." When the term "hair cells" is considered identical to "sensory cells," this is probably in some degree a result of synthetic speculation, as it is almost impracticable in these sections to determine the character of the cell from which such a "hair" arises. This difficulty is due to the circumstance that the transition of the hair in the cell itself cannot be followed, and hence it cannot be decided whether the "hair" comes from one of the luminal columnar cells or perhaps from the projecting part of a basal cell, situated behind the other.

It is no hazardous conclusion to assume that the elements mentioned in the literature as "cilia" or "sensory hairs" are identical to those here described as cytoplasmic extensions from the crista cells. In the cod, presumably, these structures are preponderantly extensions from the sensory cells (see photomicrographs), while in the shark it cannot be decided whether the basal cells, which in the fetus appear to take part in the building-up of the cupula as described here,

also take part in the formation of the cytoplasmic extensions in the adult animal.

It is surprising that previous investigators with the limited facilities at their disposal were able to enter as far into these problems as they did; and it is not to be wondered that the cytoplasmic granules on the cilia or nuclei in the subcupular zone were taken to be artefacts with the methods then employed.

#### DISCUSSION

If it may be taken for granted that the findings reported correspond on the whole to the actual structure and structural changes of the cupula, the question about the consequences of these findings suggests itself.

In the writer's opinion the entire structure described signifies the ability of nature to transmit the mechanical impulse to the sensory epithelium in the most dynamic-economical way possible, while the mechanism described presumably permits a finely graduated transmission of force. On deflection of the cupula, the subcupular zone "opens" on one side and its contents are quite readily pulled tangentially over the surface of the sensory epithelium. This effect may conceivably be brought about in two ways, which perhaps co-operate with one another. In the first place, a *traction* is exerted from the area of the "opening" of the subcupular zone; in the next place, a *pressure* is exerted on the other side from the stretched marginal bundles of fibrils. This results in a very slight shift of the contents of the subcupular zone. If the protoplasmic extensions of the sensory cells are considered as an integrant part of the sensory cells, these cells themselves may be said to be deflected or deformed. What this may give rise to is still unknown, and as yet we know nothing particular about the possible presence of hormonal intermediaries, altered potentials of the membranes, migration of ions, etc.

It appears as if the mechanism of energy exchange mentioned takes place chiefly in the subcupular zone. Probably the peripheral part of the cupula serves chiefly mechanical purposes as it transmits and perhaps accentuates the mechanical impulses from the endolymph. The interaction between the sensory epithelium and the subcupular zone as well as the interaction between this zone and the rest of the cupula probably proceeds far more intimately than could reasonably be imagined before, and presumably the sensory epithelium constitutes, together with the cupula, an inseparable and indivisible unity.

## SUMMARY

On examination of freshly extirpated cod ampullae treated by the Altmann-Gersh freezing-drying method the author has been able to demonstrate a definite structure at the site of the ampullary cupula.

This structure is the same in all the specimens, subject to regular variations in the different phases of deflection of the cupula.

Further, the writer has demonstrated the presence of a subcupular zone containing a nucleated syncytial reticulum in intimate connection with the sensory cells.

The author assumes that the peripheral fibrillary part of the cupula serves entirely mechanical purposes, and that its deflection elicits such changes in the subcupular zone as result in altered nerve action potentials.

The subcupular zone is taken to be a projecting part of the sensory epithelium itself.

In collaboration with Dohlman, it is pointed out that the motion of the cupula appears chiefly to take place as a *sliding* (on the border between the two cupular zones), while deflection takes place but to a lesser degree.

Some details of the structure of the cupula and its variation under deflection of the cupula are outlined.

UPLANDSGADE 36, B.

## REFERENCES

1. Altmann, R.: Die Elementarorganismen und ihre Beziehungen zu den Zellen, Leipzig, 1894.
2. Bowen, R. E.: The Cupula of the Membranous Labyrinth, J. Comp. Neurol. 58:517, 1933.
3. Buchanan, F.: On the Time Taken in Transmission of Reflex Impulses in the Spinal Cord of the Frog, Quart. J. Exper. Physiol. 1:1, 1908.
4. Dohlman, G.: Film showing the movements, etc., of injected cupulae, 1928, and personal communication with information on his unpublished experiments.
5. Gersh, J.: Altmann Technique for Fixation by Drying While Freezing, Anat. Rec. 53:309, 1932.
6. Hoerr, N. L.: Cytological Studies by Altmann-Gersh Freezing-Drying Method, Anat. Rec. 65:293, 1936.
7. Kolmer, W.: Studien am Labyrinth von Insectivoren, Sitzber. d. Kaiserl. Akad. d. Wiss. Vienna, Math. natw. Kl. 122:29, 1913.
8. Löwenstein, O., and Sand, A.: The Individual and Integrated Activity of the Semicircular Labyrinth, J. Physiol. 99:89, 1941.
9. Möllgaard, H.: Die vitale Fixation d. Zentralnervensystems, Wiesbaden, Copenhagen, 1911.

10. MacNally, W., and Tait, J.: Some Results of Section of Particular Nerve Branches to the Ampullae of the Vertical Semicircular Canals of the Frog, *Quart. J. Exper. Physiol.* 23:147, 1933.
11. Sjöstrand, F.: Ueber die Eigenfluoreszenz tierischer Gewebe, Stockholm, 1944.
12. Steinhausen, W.: Ueber die Beobachtung der Cupula d. Bogengangsampullen d. lebenden Hechts, *Pfl. Arch. d. ges. Physiol.* 232:500, 1933.
13. Vilstrup, Th.: Studies on the Histogenesis of the Ampullary Cupula, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 59:19, 1950.
14. Werner, Cl. F.: *Das Labyrinth*, Leipzig, 1940.
15. Glimstedt, G.: Personal communication.
16. Simpson, W. L.: Experimental Analysis of Altmann Technique of Freeze-drying, *Anat. Rec.* 80:173, 1941.
17. Retzius, G.: *Das Gehörorgan. d. Wierbeldiere*, Stockholm, 1884.
18. Wittmaack, K.: Ueber d. Tonus der Sinnesendstellen des Innenohres, *Arch. f. Ohren.-Nas.-Kehlkopfheilkunde* 114:278, 1926.

#### IV

### THE PHYSIOLOGY OF RESPIRATORY OBSTRUCTION

JOHN S. GRAY, M.D.

CHICAGO, ILL.

Obstruction of the upper respiratory passages occurs in a variety of diseases and conditions ranging from the acute laryngotracheobronchitis of children, through foreign bodies and tumors, to any of those states in which the self-cleansing mechanism of the respiratory passages are depressed, paralyzed or interfered with. Such obstructions potentially carry a grave threat to the patient's life, since they may abruptly pass from an apparently compensated stage to a fulminating and fatal asphyxia. The laryngologist is confronted with the treacherous problem of gauging the present status of the patient's condition and predicting its future course, as a basis for deciding between conservative management and tracheotomy. Any addition to his knowledge and understanding of the underlying physiopathology of obstructive states would presumably be welcome as an aid in improving his judgment in these trying circumstances. It is on that basis no doubt that I have been asked to discuss the physiology of respiratory obstruction.

Let us begin with a simple obstruction, uncomplicated by accompanying lung pathology. This is more convenient for purposes of analysis, and it can be used as a nucleus to which the additional effects of complications can be added later. Our simple obstruction is located above the bifurcation of the trachea, so that the entire lung shares uniformly in the interference produced by the obstruction.

The very first physiological change resulting from this obstruction will be a reduction in the *ventilation capacity* of the pulmonary bellows. Healthy men on the average can breathe about 170 liters of air per minute when they voluntarily breathe as hard as possible.<sup>1</sup> This is the normal ventilation capacity. In respiratory obstruction this capacity is affected early and potentially to a very marked degree. For example, experiments on healthy subjects have shown that an artificial obstruction consisting of an orifice with a diameter one-

---

From the Department of Physiology, Northwestern University Medical School, Chicago, Ill.

Read before the meeting of the Chicago Laryngological and Otological Society, Chicago, Illinois, November 7, 1949.

half that of the trachea reduces the ventilation capacity to about 128 l/min., or to 75% of the normal.<sup>2</sup> If the orifice diameter is only one-fourth that of the trachea, the ventilation capacity falls to about 40 l/min., which is only 25% of the normal. The latter is a real impairment of the ability to breathe and is comparable to a moderate to severe asthma or obstructive emphysema. The earliest effect of the obstruction, therefore, is to impair the ventilation capacity, which if mild means dyspnea on exertion and if severe means dyspnea even at rest.

The second respiratory disturbance which the obstruction will produce is a condition known as *hypoventilation*. Normal persons under resting conditions breathe on the average about 7.0 l of air per minute. This is the resting pulmonary ventilation, or respiratory minute volume. Under these same conditions, about 275 cc of O<sub>2</sub> are consumed per minute. The important ratio between these two is known as the ventilation equivalent for O<sub>2</sub> and has a normal value at rest of about 2.5.<sup>3</sup> This means that 2.5 l of air are breathed for each 100 cc of O<sub>2</sub> absorbed. If the O<sub>2</sub> consumption is increased, for example, by hyperthyroidism, or by moderate exercise, this ratio remains unchanged, because the pulmonary ventilation normally keeps pace with the increased O<sub>2</sub> exchange.<sup>4</sup> In respiratory obstruction, however, this relationship is broken and the ventilation equivalent for O<sub>2</sub> tends to fall. In the early stages this will occur only on exertion when the ventilation, because of respiratory resistance, is unable to keep pace with the increased O<sub>2</sub> consumption. In later stages, however, the ratio will fall even at rest, partly because of the increased O<sub>2</sub> consumption necessary to support the extra respiratory effort, and partly because the resistance may reduce the ventilation itself below normal. The reduced ventilation equivalent for O<sub>2</sub>, therefore, betrays the failure of ventilation to meet body requirements, a condition called hypoventilation.

As hypoventilation becomes more and more severe it induces progressive chemical changes in the blood which may have disastrous consequences for the patient. First of all, the hypoventilation causes a retention of CO<sub>2</sub> in the body with a resulting increase in the blood CO<sub>2</sub> content, especially of free CO<sub>2</sub>. This is known as *hypercapnia* and is measured by the rise in the arterial CO<sub>2</sub> tension. Secondly, hypoventilation brings about a fall in the O<sub>2</sub> saturation of the arterial blood, a condition properly called an arterial *anoxemia*. Thirdly, both the hypercapnia and the anoxemia produce an *acidemia* or fall in the blood pH. The increased free carbonic acid of the blood lowers the pH in proportion to the excess carbonic acid. This is a respiratory acidosis since it is due to respiratory interference with

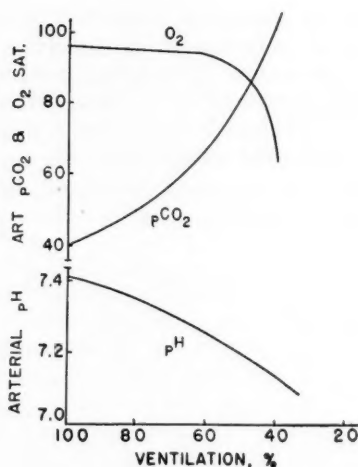


Fig. 1.

CO<sub>2</sub> elimination through the lungs. If the anoxemia becomes severe, the O<sub>2</sub> starved tissues will produce excess lactic acid, the accumulation of which in the blood constitutes a metabolic acidosis. Now if both a respiratory acidosis due to volatile carbonic acid, and a metabolic acidosis due to fixed lactic acid are present simultaneously, neither one can be compensated. The result, therefore, is a potentially severe form of acidosis.

This trinity of blood changes, comprising hypercapnia, anoxemia, and acidemia, is the inevitable consequence of hypoventilation.<sup>5</sup> It is properly called *asphyxia*, a word which implies interference with both O<sub>2</sub> and CO<sub>2</sub> exchanges. It is not simple anoxia, for this is ordinarily accompanied by hyperventilation, acapnia, and alkalemia. It is something far worse than simple anoxia, because the retained CO<sub>2</sub> makes a very important contribution to the disastrous effects of asphyxia as we shall shortly see.

But before describing the effects of asphyxia, it will be well to inquire into the magnitude of the changes in blood composition in relation to the degree of hypoventilation. Figure 1 illustrates such changes as they would occur in a simple, uncomplicated respiratory obstruction. In this figure the horizontal axis represents the adequacy of the pulmonary ventilation expressed in percentage of normal. The vertical axis is divided into two scales; the upper scale applies to the arterial O<sub>2</sub> saturation in per cent and the CO<sub>2</sub> tension in mm of Hg, whereas the lower scale refers to the arterial pH.

With normal ventilation, i.e., 100%, the  $\text{CO}_2$  tension is at 40 mm of Hg, the  $\text{O}_2$  saturation at 96% and the pH at 7.41. With moderate hypoventilation, say at 80% of normal, the  $\text{O}_2$  saturation is not detectably affected, but the  $\text{CO}_2$  tension rises to 50 mm of Hg and the pH falls to 7.35, both of which are definitely beyond the normal ranges. This predominance of hypercapnia over anoxemia has been noted in experimental respiratory obstruction in dogs.<sup>6</sup> If the hypoventilation progresses to 50% of normal, the  $\text{O}_2$  saturation falls to about 88%. This anoxemia is equivalent to that produced by exposure to an altitude of less than 10,000 feet, which is readily tolerated, and which does not produce cyanosis. The  $\text{CO}_2$  tension, however, has reached 80 mm of Hg, which approaches the narcotic level for this gas. The pH has fallen to 7.20 which is a real acidosis, but due exclusively to  $\text{CO}_2$  retention, since the anoxemia is still too mild to produce lactic acidosis. Further restriction of ventilation brings precipitous changes in all three blood constituents, as the graph shows.

This figure applies to simple, uncomplicated respiratory obstruction. Two important modifications are produced by pulmonary complications such as infection, edema, scattered areas of atelectasis, and partial blocking of scattered bronchi or bronchioles, which are apt to accompany clinical obstructions. The first modification is that the arterial  $\text{O}_2$  saturation will be lower than indicated in the graph, and the second is that the pH will likewise be lower because of the earlier appearance of lactic acidosis in the presence of anoxemia. Pulmonary complications, therefore, intensify the anoxemia and acidosis.

It has been traditional to attribute all the adverse effects of this trinity of blood changes to one of them alone, namely, the anoxemia. In fact, these changes are often described as anoxia. There is no doubt that severe anoxemia can be and often is fatal. But one cannot afford to neglect the equally serious effects of severe hypercapnia and acidemia. For example, the distress, apprehension, restlessness, disorientation, and unco-operativeness of a patient with respiratory obstruction are probably more apt to be due to hypercapnia than to anoxemia. It appears not to be generally recognized that hypercapnia is as distressing as anoxemia is symptomless. A rapidly produced simple anoxia in which the arterial  $\text{O}_2$  saturation falls below 60% may produce unconsciousness without distress, or sensation, or even any premonition of danger. This has been demonstrated literally thousands of times to Air Force personnel during the last war. In marked contrast, breathing 10%  $\text{CO}_2$ , which elevates the arterial  $\text{CO}_2$  tension to about 60 mm of Hg without any accompanying anoxemia, produces extreme distress, apprehension, fear of impending

suffocation and can be tolerated only for about 15 minutes. Longer exposures and higher concentrations induce narcosis, anesthesia, respiratory depression, and circulatory collapse.<sup>7</sup> The potential danger of acidosis is probably better understood than that of hypercapnia, since everyone is familiar with the coma which accompanies diabetic acidosis when the pH falls to 7.0 or 7.1. In asphyxia, therefore, the threat of anoxemia is bad enough, yet it represents only one-third of the potential danger.

In the earlier stages of respiratory obstruction, these asphyxial blood changes provide a powerful respiratory stimulus which aids in the maintenance of breathing in the face of the obstructive resistance. The hypercapnia, acidemia, and anoxemia are each powerful respiratory stimuli<sup>8</sup> and together they bring into play the various accessory muscles of respiration. If the obstruction and resulting hypoventilation continue to progress, however, there comes a point of *crisis* where the asphyxial blood changes cease to behave as respiratory stimuli, and the breathing effort weakens. Because of this failure of respiratory effort, the asphyxial changes are intensified, and breathing is then actually inhibited. The result is a vicious cycle, a fulminating and self-accelerating asphyxia which may terminate abruptly and fatally.

Let us examine the mechanism of this crisis and the resulting sudden respiratory failure. Carbon dioxide in moderate concentrations is a powerful respiratory stimulus, but in high concentrations, its effect is reversed and transformed to inhibition. To make matters worse, the concentration at which this CO<sub>2</sub> reversal takes place is lowered if the respiratory centers are depressed for any reason. In severe asphyxia the respiratory centers do become depressed by the progressive anoxemia and acidemia (a situation which will only be aggravated by the use of sedatives). As a result the previously effective respiratory stimuli now become ineffective, and CO<sub>2</sub> actually becomes inhibitory. As the breathing effort weakens these adverse effects are intensified. The vicious cycle then brings about rapid termination of breathing.

If the crisis of weakening respiratory effort is reached, there may be only minutes left in which successful intervention is possible. Furthermore, the crisis may appear so abruptly that it is dangerous to allow events to progress too far under conservative management. In fact, if the management is to be conservative of the patient's life, decisive intervention may be necessary.

Although O<sub>2</sub> therapy is important and wholly beneficial in the treatment of respiratory obstruction, it must never be forgotten that

it cannot correct all the asphyxial changes.  $O_2$  therapy will relieve the arterial anoxemia and thus supply the tissues with the necessary  $O_2$ . This will aid the respiratory centers in withstanding the inhibitory effects of excessive  $CO_2$ , and will also correct that portion of the acidosis which is due to lactic acid accumulation. However important these benefits are,  $O_2$  therapy cannot correct the hypercapnia, nor its contribution to the acidosis. Nor will  $O_2$  therapy relieve or prevent the development of pulmonary complications which are secondary to the obstruction. Only relief of the respiratory obstruction and maintenance of the airway can reverse these and other effects of respiratory obstruction.

This analysis of the underlying physiopathological changes implies that any respiratory obstruction carries with it the potentiality of reaching the critical phase of weakening respiration and fulminating asphyxia. Perhaps this analysis will serve to strengthen the laryngologist in his conviction that careful observation, experienced and skilled judgment, and decisive action are paramount in the successful management of respiratory obstruction.

#### REFERENCES

1. Gray, J. S., Barnum, D. R., Matheson, H. W., and Spies, E. N.: Ventilatory Function Tests. I. Voluntary Ventilation Capacity. To be published.
2. Matheson, H. W., Spies, S. N., Gray, J. S., and Barnum, D. R.: Ventilatory Function Tests. II. Factors Affecting the Voluntary Ventilation Capacity. To be published.
3. Matheson, H. W., and Gray, J. S.: Ventilatory Function Tests. III. Resting Ventilation, Metabolism, and Derived Measures. To be published.
4. Gray, J. S.: Pulmonary Ventilation and Its Physiological Regulation, Springfield, Charles C. Thomas. To appear in 1949 or 1950.
5. Grodins, F. S., Lein, A., and Adler, H. F.: Changes in Blood Acid-Base Balance during Asphyxia and Resuscitation, *Am. J. Physiol.* 147:433, 1946.
6. Blalock, A., Harrison, T. R., and Wilson, C. P.: Partial Tracheal Obstruction; Effects on Circulation and Respiration of Morphinized Dogs, *Arch. Surg.* 13:81, 1926.
7. Seevers, M. H.: Narcotic Properties of Carbon Dioxide, *N. Y. State J. Med.* 44:597, 1944.
8. Gray, J. S.: The Multiple Factor Theory of the Control of Respiratory Ventilation, *Science* 103:739, 1946.

V

RADIUM THERAPY FOR LYMPHOID TISSUE IN THE  
NASOPHARYNX

EDWIN B. BILCHICK, M.D.

AND

ALBERT R. KOLAR, M.D.

NEW YORK, N. Y.

This article deals with the use of radium in the nasopharynx for the prevention and treatment of otic, nasopharyngeal and nasal complaints. Treatment of neoplasms is excluded in this discussion. The material consisted of a total of 468 cases from the Otolaryngological Department of the Columbia-Presbyterian Medical Center, including the Vanderbilt Clinic. Some of the private cases of Dr. Fowler and one of the authors (E. B. B.) were available for this study. Out of the total number (468) there were available 253 records, but not all were complete in all respects so that detailed analysis of each case could not be obtained nor were follow-up data on all patients obtained. However, the series enabled us to draw some definite conclusions concerning the use of radium in the nasopharynx.

The use of radium for deafness associated with hypertrophied nasopharyngeal lymphoid tissue and for allied ailments has been pioneered by the Johns Hopkins Hospital group headed by Crowe.<sup>1</sup> Fowler,<sup>2-4</sup> assisted by the Columbia University Radiation and Physics Department has also contributed to this work which was enhanced by his experiences in England while treating aero-otitis. The earlier reports largely concerned the use of radon whereas our present comments involve mainly the use of the 0.3-mm Monel metal applicator standardized by Crowe for the Army Air Force. After a few years of work with his standard technique he has extended the time of treatment from  $6\frac{3}{4}$  minutes to 12 minutes, since it has been determined that the radium salt in the applicator absorbs a certain amount of the active radiation.<sup>5</sup> The time interval between treatments has been from two to four weeks.

It has been found in a number of our cases that following use of the applicator in the nasopharynx there has been a tendency for

---

From the Department of Otolaryngology, Columbia-Presbyterian Medical Center.

otitis media to flare up on the eleventh to the thirteenth day, indicating height of the local erythema. Because of these considerations we favor the use of four treatments for 9 minutes at three-week intervals. On the other hand we have given a number of cases 12-minute applications at intervals of two weeks to evaluate the result of this regime and because occasionally only four weeks are available for treatment. Crowe and his co-workers claim no reaction to the irradiation as a rule. It is possible that our incidence of reaction with the higher dosage is due to the fact that we always shrink the turbinates before application; we always surgically remove excess adenoid tissue on the posterior nasopharyngeal wall and we cross our applicators so that the radium capsule is in close approximation with the excessive lymphoid tissue at the eustachian orifice.

For delivery of the calculated dosage the side of the applicator should be exactly on the edge of the eustachian tube. Variations in the depth and width of the nasopharynx, in the size and degree of shrinkage of the inferior turbinates, the presence of narrow nares and septal deviations make exact placement impossible, especially in small children. A rigid straight applicator in practice can generally only be passed straight in along the floor of the nose as laterally as possible. An applicator with a curved tip was designed to insure better approximation, but it often fell out of place and could not be used. It is therefore likely that the calculated dosage is the maximum delivered and in most cases actual delivered dosage is less than the calculated dose, for the original calculations were made for radon rather than radium. According to Crowe and his co-workers 2 gm-min. with the brass radon applicator is equivalent to 18 minutes with the 50-mg Monel metal applicator. Much discussion has arisen concerning the actual dose delivered by the 50-mg, 0.3-mm Monel metal applicator. Dr. Quimby, our radiation physicist, states that up to the present time no careful physical measurements of the actual radiation doses have been made.\* She has made a very rough estimate of the dosage, as follows: Radium filtered by 0.3 mm Monel metal delivers 75% beta and 25% gamma. The 50-mg applicator used for  $6\frac{2}{3}$  minutes should deliver 1500 r at a depth of 1 mm in tissue; in  $12\frac{1}{2}$  minutes, 2860 r. At a depth of 1 cm the dose is approximately 50 r.

Irritation from the radium applicator may develop 24 to 48 hours after treatment, but not uncommonly there may also be throat irritation 8 to 13 days after application. The patient may complain of a sandy or painful throat, with intermittent radiation of pain to

\*Measurements of this type are currently being done by Mr. Carl Braestrup of the New York City Department of Hospitals. These should be available by the end of 1949.

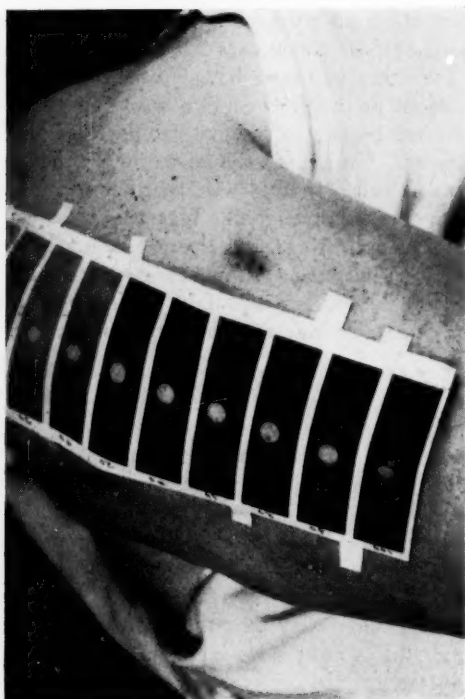


Fig. 1.—Skin reaction on outer arm after 12-minute radium application.

the ears. Examination at this time reveals pharyngeal redness, with some edema extending to the nasopharynx. These early and late reactions to radium are also seen on the skin when radium is directly applied for testing skin sensitivity. A number of cases were tested with the standard applicator and observations taken at intervals revealed burning sensation, edema and redness, and pigmentation lasting six weeks. Figure 1 shows the skin reaction on the upper outer arm photographed 12 days after a 12-minute exposure. When compared with the standard Tallquist hemoglobin scale the color reaction approximates the 70% hemoglobin level. Although this does not exactly indicate the degree of damage and amount of reaction produced when applied in the nasopharynx, it reminds us of the potency of the agent in use.

Radium causes marked alterations of the surface epithelium and lymphoid tissue of the nasopharynx. Schenck<sup>6</sup> in 1941 described

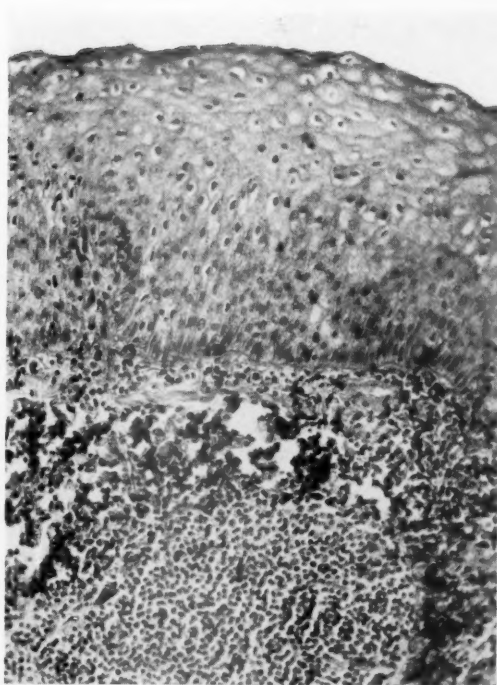


Fig. 2.—Photomicrograph of section of nasopharynx following radium therapy. Low power shows thickening of epithelium, activity in basal layer, mitotic figures in great numbers, and distortion of germinal centers.

the histological findings in tissue removed from 22 children aged 6-12 before and after irradiation. He used x-ray, radium and a combination of the two and found swelling of the ciliated cells with detachment of these cells two to four weeks after irradiation. There was a marked effect on the lymph capillary endothelium with destruction and contraction. Many cells were liquefied. After six months germinal centers had not appeared, with identical results regardless of modality employed. Fowler<sup>4</sup> described sections of lymphoid tissue taken four weeks after 50 mg-hr. exposure to radium in a platinum capsule with 0.5-mm wall. He noted absence of germinal centers and prominence of the capillary endothelium, with increase of connective tissue. Fowler<sup>7</sup> also describes a radium reaction in a young man who had had previous x-ray and radium irradiation and later received a single 9-minute treatment with the standard applicator. He developed a bluish red appearance of the

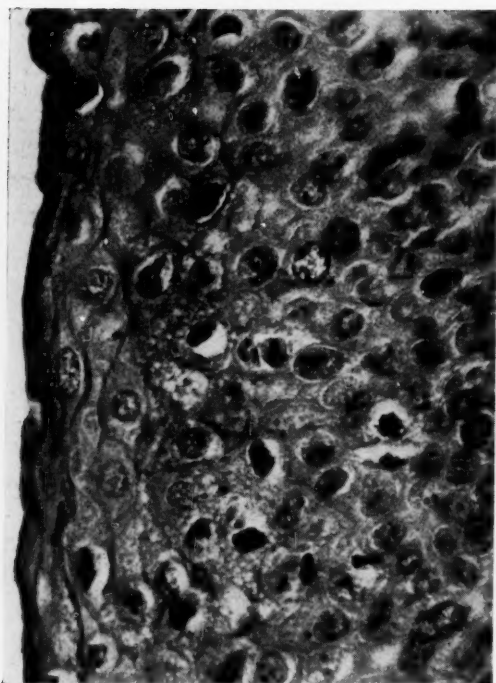


Fig. 3.—Photomicrograph of high power view of epithelium shown in Fig. 2. Large number of mitotic figures are seen.

entire nasopharynx and upper part of the pharynx which lasted a month. Biopsy taken four weeks after the last treatment showed such numerous mitotic figures in the epithelium that the pathologist considered the possibility that it might be the onset of an intramucosal carcinoma.

Because of the possibility of reactions just described, careful questioning of the patient and examination of the throat and nasopharynx when feasible have been carried out prior to each treatment. When patients complain of a recent cold or dryness or irritation of the throat, or when signs of throat irritation or acute infection are found, treatment has usually been postponed until cessation of symptoms and signs.

To further study the effect on the surface epithelium a biopsy was taken from a 41-year-old patient who had received irradiation. This man had complained of clogging of the right ear and poor hear-

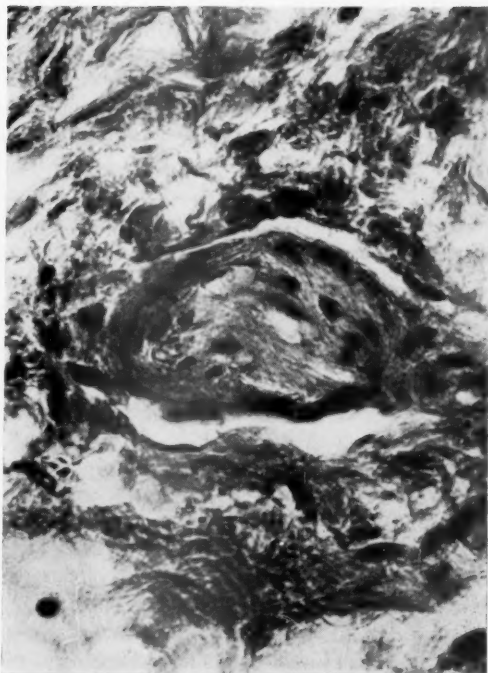


Fig. 4.—Photomicrograph of high power view of subepithelial layer showing thickened hyalinized capillaries and prominent endothelial cells.

ing for three months, not relieved by local nasal treatments or inflations. A secondary tonsillectomy and adenoidectomy had been done for tonsillitis on November 11, 1937. Audiometric tests showed good hearing in both ears. Lymphoid tissue was found around the tubes. The sinuses were clear. On December 15, 1948, he received a 10-minute treatment to the nasopharynx with the 50-mg applicators. On January 5 and January 26, 1949, he received 12-minute treatments, but his complaints did not subside and he noticed bleeding from his nose. Nasopharyngoscopic examination showed continued presence of engorged lymphoid tissue around the tubes. On February 21, 1949, under ether anesthesia the nasopharynx was explored and the remaining lymphoid tissue removed with curettes and punches.

Histological examination was reported as follows: "The sections show marked hyperplasia and distortion of the epithelial cov-

ering. The epithelium is hyperplastic, contains many mitoses and is markedly disoriented and disrupted, with infiltration of inflammatory cells. The basal layer shows evidence of moderate activity. Many strands of squamous epithelial cells dip into the lymphoid tissue surrounding it. Underneath the epithelium are thickened areas of fibrosis, infiltrated with many chronic inflammatory cells. In this tissue many thick walled arterioles are seen. The capillaries show thickened walls. In many of the areas there is alteration of the germinal follicles, some of which frequently consist of only a few large reticulum cells." (Worcester) (Fig. 2, 3 and 4.)

Studies of these sections and those of Dr. Fowler impress us with the effect of irradiation on the nasopharyngeal epithelium as well as the lymphoid tissue. Proliferation of epithelium often occurs with chronic infection, but we always wonder whether it might not become abnormal after irradiation and result some day in epithelioma. Herein lies the principal danger of the treatment. So far no serious complication has been reported from any clinic.

It is, of course, necessary to avoid accidents due to technical difficulties such as breakage of applicator wires, leaking radium chamber, mistake in time of treatment, excessive dosage or repeated courses of treatment without sufficient interval. The dangers of even properly calculated treatment have been emphasized by some writers. Schulz and Robbins<sup>8,9</sup> of the Department of Radiology, Massachusetts General Hospital, have issued a stern warning concerning the use of the present treatment. They state that the dosage is too high and that there is danger of long-delayed tissue destruction. However, they admit no experience with the nasopharyngeal applicator under discussion. They describe the deleterious late effects of radium and radiotherapy in general when used in other areas such as skin and bony tissue, citing lesions arising as long as 23 years later, with ulcerations, hyperkeratoses, atrophies, bone necroses and malignant changes. They have attempted to compare the estimated dosage given in these cases with the estimated similar dose of the 50-mg radium capsule. But they take no cognizance of the fact that comparing large-field x-ray dosage with that from the tiny applicator is probably not valid. After a lengthy comparison of estimated dosage of the radium with x-ray, they admit that the exact output of this applicator is still "indefinite and incalculable." On the other hand, Crowe<sup>10</sup> stated in a symposium on this subject that for several years he has "supplemented every tonsillectomy with irradiation of the nasopharynx." How can we reconcile these seemingly conflicting statements? We believe the safest and most practical stand to lie between these two extreme viewpoints. Although

Crowe's group has used irradiation for 25 years with no report of complication it cannot be safely assumed that no ill effects will occur in the future and we must be prepared for them. No one can predict when an evidently increased proliferative mitotic process may break through the basement membrane and become a neoplastic growth. In the course of time, by the law of average there will develop a certain number of neoplasms of the nasopharynx, some in patients who never received irradiation, some in patients who did receive irradiation. A scientific unhysterical attitude will then be needed to evaluate such a situation in its true light. And if any of the patients who receive it or any of the physicians who use the radium develop agranulocytosis or anemia a complete investigation and reappraisal of the subject will be in order.

The patients selected for treatment in our series were those who complained of otic, nasopharyngeal or posterior nasal symptoms. Treatments were not limited to patients with deafness and an attempt was made to analyze and treat various groups of symptoms in order to determine the relative therapeutic value in these groups. Some cases were treated even though the lymphoid tissue was minimal. In a few cases treatment was begun in the presence of large masses of lymphoid tissue. It was soon evident that no improvement was forthcoming, so surgical adenoidectomy was performed. The commonest symptoms recorded were, in order of frequency: loss of hearing, tinnitus and vertigo, recurrent ear and throat infections, stuffy nose, postnasal discharge, asthma. Objective findings, aside from the presence of lymphoid tissue in the nasopharynx, were principally recurrent conductive deafness, high tone deafness, chronic suppurative otitis media. Cases with high tone losses suggesting definitive nerve involvement were occasionally treated because it was felt that there might be an added conductive deafness which could be reduced by the therapy. In general these cases were treated only when they reported fluctuation in hearing with colds.

In estimating the results of treatment it was found convenient to separate the cases into two age groups: children up to 15 and those over 15. Of 104 children who comprised the first group 68 complained of deafness which was shown in the initial audiogram in 57 cases. Among this group were 22 cases of nerve deafness, 35 cases of conductive deafness. In another group of 30 children complaining of colds, coughs or discharging ears, there were 13 cases of conductive deafness, making a total of 48 cases of conductive deafness.

After treatment of the cases of nerve deafness only one was found to be improved. Treatment of cases of conductive deafness, however, resulted in substantial improvement in 37 of the 48 cases.

But the general picture of the other complaints such as tinnitus and recurrent discharging ears was not remarkably altered by the irradiation, with few exceptions.

Of 148 cases over 15 years of age, initial objective deafness was found in 102. Of these, 72 were cases of conductive deafness, 30 of nerve deafness. Only 2 of the cases of nerve deafness were improved by treatment, whereas 36 of the cases of conductive deafness were improved. Of the cases complaining of discharging ears, tin-

TABLE 1.  
RESULTS\* OF RADIUM TREATMENT OF CASES OF OBJECTIVE  
DEAFNESS

AGE	TOTAL NUMBER	IMPROVED		UNIMPROVED	
		NO.	%	NO.	%
2-15	70	37	52.8	33	47.2
15-70	102	38	37.2	64	62.8

TABLE 2.  
RESULTS\* OF TREATMENT OF CONDUCTIVE DEAFNESS

AGE	NUMBER	IMPROVED		UNIMPROVED	
		NO.	%	NO.	%
2-15	48	37	77	11	23
15-75	72	36	50	36	50

\*Improvement equals more than 10 db for 500, 1000, 2000 averaged together.

nitus, vertigo or postnasal discharge, only an occasional case seemed improved. The notes on the records were inadequate in the individual cases to substantiate this impression for statistical purposes. Except in cases presenting marked initial hypertrophy, visible decrease in lymphoid tissue was equivocal in most cases, for residual small amounts were commonly seen. Dryness of the throat, irritation of the throat, flare-up of previous otitis media were seen in almost half of the patients who were closely followed and queried as to postradiation reaction.

X-ray therapy has been used for deafness and allied complaints prior to the use of radon and radium. X-ray therapy is preferable when the lymphoid tissue extends beyond the eustachian tube orifice, when the pathology involves the middle ear or extends below the nasopharynx into the pharynx and causes symptoms. Fowler<sup>11</sup> in 1942 reported treatment of 63 cases with x-ray. His patients were frequently relieved of clicky and stuffy ears. Colds and recurrent otitis were reduced and stuffy noses were helped. The hearing losses were frequently reduced, especially recent small losses. The x-ray dosage was 75-100 r per sitting twice a week until 1000 r to each side was given. Youngs and Woutat<sup>12</sup> recently reported treatment of 116 cases of deafness with 125-150 r to each of two 4-5-cm ports on each side of the face, using four weekly treatments. They obtained great improvement in most cases, with only minimal occasional transient swelling and discomfort over the parotid region.

Review of some of our patients who received radiotherapy indicates some improvement, but not such a high percentage as in the radium group. Undoubtedly, although no bad results have been reported, radiotherapy distributed over a larger area through numerous tissues including skin, involves a greater risk than radium therapy limited to a small area. Radium is easier to use, does not require such massive equipment and does not tend towards parotid irritation and dryness of the pharynx. Both methods of therapy have their place, and continued comparisons will eventually determine their relative efficacy and safety.

Caution is needed lest we abandon other effective means of therapy in the treatment of ear conditions and make the use of radium a routine procedure, which it should never become. The tendency to omit complete examination and treatment of the nose and sinuses and allergic work-up and simply order radium therapy must not be countenanced. Inflation of the eustachian tubes, myringotomy and adenoidectomy must not be omitted when needed. To neglect the use of antibiotics and chemical agents and vasoconstrictors which by themselves can frequently cure a case and prescribe radium as a short-cut will not further the reputation of radium as a therapeutic agent. In one particular condition it would be foolhardy to rely entirely on radium: that is secretory otitis media. Persistent fluid in the middle ear with hearing impairment should be treated by myringotomy and suction-aspiration of the contents of the middle ear.<sup>13</sup> This may have to be repeated. Radium should be considered not as a panacea but as one method of treatment, which, when used in conjunction with other therapeutic methods, will achieve the best results.

## SUMMARY

1. Radium therapy of the nasopharynx to reduce lymphoid hyperplasia is an excellent effective method of prevention and therapy for conductive deafness in children. A high proportion of a group of children who received this treatment was greatly benefited.

2. A large proportion of adults with conductive deafness was greatly benefited by this treatment, but the percentage of improvement is less than when employed in children.

3. Irradiation has no effect on nerve deafness, but may have an effect on a concomitant conduction element.

4. Allied symptoms involving the ears, nose or throat were benefited in only a few cases.

5. The destructive effects on lymphoid tissue and the proliferative effects on epithelium with its possible dangers are described.

6. Caution should be exercised in the use of radium, in selection of cases and technique of application. Special danger is incurred by repeated series of treatments.

Mrs. Frieda D'Amato, A.B., M.A., rendered valuable assistance with the hearing tests and analyses.

133 EAST 58TH STREET.  
640 PARK AVENUE.

## REFERENCES

1. Crowe, S. J., and Burnam, C. F.: Recognition, Treatment and Prevention of Hearing Impairment in Children, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 50:15-31 (Mar.) 1941.
2. Fowler, Edmund P., Jr.: Causes of Deafness in Flyers, *Arch. Otolaryng.* 42:21-32 (July) 1945.
3. Fowler, E. P., Jr.: Use of Radon to Prevent Otitis Media Due to Hyperplasia of Lymphoid Tissue and Barotrauma (Aero-Otitis), *Arch. Otolaryng.* 40:402-405 (Nov.) 1944.
4. Fowler, E. P., Jr.: Irradiation of the Eustachian Tube, *Arch. Otolaryng.* 43:1-11 (Jan.) 1946.
5. Proctor, D. F., Polvogt, L. M., and Crowe, S. J.: Irradiation of Lymphoid Tissue in Diseases of the Upper Respiratory Tract, *Bull. Johns Hopkins Hosp.* 83:383-428 (Nov.) 1948.
6. Schenck, Harry P.: The Influence of Nasopharyngeal Hyperplasia on the Ear. Histologic Examination of Hyperplastic Lymph Follicles after Irradiation, *Laryngoscope* 51:780-789 (Aug.) 1941.
7. Fowler, E. P., Jr.: Eustachian Tube Irradiation, *N. Y. State Med. J.* 49:187-190 (Jan. 15) 1949.
8. Schulz, M. D., and Robbins, L. L.: Dangers of Irradiation of Hypertrophied Lymphoid Tissue of the Nasopharynx, *Tr. Am. Acad. Ophth. and Otolaryng.* 53:243-252 (Jan.) 1949.

9. Robbins, L. L., and Schulz, M. D.: Potential Hazards from Radiation Treatment of Hypertrophied Lymphoid Tissue in the Nasopharynx, *Laryngoscope* 59:147-155 (Feb.) 1949.
10. Crowe, S. J.: Irradiation of the Nasopharynx, *Tr. Am. Acad. Ophth. and Otolaryng.* 51:29-35 (Sept.) 1946.
11. Fowler, E. P., Jr.: Non Surgical Treatment for Deafness, *Laryngoscope* 52:204 (Mar.) 1942.
12. Youngs, N. A., and Woutat, P. H.: Treatment of Certain Types of Deafness by Roentgen Ray, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:984-991 (Dec.) 1948.
13. Tobey, H. G.: *Tr. Am. Otol. Soc.* 36:51-64, 1948.

## VI

### RADIATION EXPOSURE OF PERSONNEL HANDLING THE MONEL METAL NASOPHARYNGEAL RADIUM APPLICATOR

HENRY J. RUBIN, M.D.

BARNEY M. KULLY, M.D.

AND

RAYMOND D. FINKLE, PH.D.

LOS ANGELES, CALIF.

The value of the 50-mg Monel metal radium applicator in the treatment of certain benign nasopharyngeal and aural conditions is recognized, and its use is now an established otologic procedure. This form of therapy is accompanied by risks inherent in the use of radium and is potentially dangerous both to the patient and to the physician and his staff. Schulz and Robbins,<sup>1</sup> in discussing dangers to the patient, emphasized the long latent period which may elapse following radiation therapy before permanent tissue damage becomes evident and suggested that insufficient time has elapsed to permit accurate evaluation of end results.

This investigation is not concerned with the patient but has been undertaken to determine the amount of radiation to which the otologist and his assistants are exposed and to evaluate the safety of that exposure in the light of present knowledge. Little data on this matter has appeared in the literature, most authorities merely warning against improper use of the nasopharyngeal radium applicator.

*Radiation Tolerance.* Although unceasing investigative work is in progress to ascertain the biologic effects of ionizing radiation, there is no unanimity of opinion as to how much irradiation the human body will tolerate without suffering permanent ill effects. That exposure to presumably safe doses could result in serious sequelae was known early, but it was not until 1922 that the American Roentgen Ray Society<sup>2</sup> made the first generally accepted proposals for radiation protection in this country. The U. S. Advisory

---

From the Departments of Otolaryngology and Radiation Therapy, Cedars of Lebanon Hospital, Los Angeles, Calif.

Committee on X-ray and Radium Protection<sup>3</sup> in its first report in 1931 suggested a body tolerance dose of 200 milliroentgens (mr) per day, and in its revised report<sup>4</sup> in 1936 it reduced this to 100 mr per day. The Standardization Committees of the American Roentgen Ray Society and the Radiological Society of North America<sup>5</sup> in 1946 also recommended 100 mr per day. Accumulating data, however, have suggested that certain biologic and genetic changes take place even at this level. Henshaw<sup>6</sup> demonstrated a shortening of the life span of mice exposed to repeated small doses of x-ray. Lorenz et al<sup>7</sup> of the National Cancer Institute showed that long continued absorption of penetrating radiation, even as low as 110 mr per day, caused a decrease in the life span of mice and a higher and earlier incidence of ovarian tumors. Sievert,<sup>8</sup> on the basis of mass blood studies in individuals working with ionizing radiation, felt that the safety tolerance dose for general irradiation should be 10 mr per day. These and other reports<sup>9</sup> of damage from small amounts of ionizing radiation led to an even keener awareness of its insidious effects. As a result the Bureau of Standards<sup>10</sup> now defines the maximum total dose to which any part of the body of a person shall be permitted to be exposed continuously or intermittently as 300 mr per week. The Subcommittee on Permissible External Dosage of the National Committee for Radiation Protection<sup>11</sup> has likewise lowered the recommended exposure value from 500 mr to 300 mr per week for gamma radiation. It will be noted that the general trend in tolerance recommendations is consistently and unmistakably downward.

There is some feeling among competent observers that a local part of the body such as the hands and fingers can tolerate higher dosage for over long periods without manifesting any untoward local or systemic effects. Well controlled experimental and clinical studies on this phase of the tolerance problem, however, are virtually nonexistent, and opinions are to a large extent based on indirect evidence. In order to determine what might be considered safe practice in regard to radiation exposure of the hands and fingers, we wrote to a number of prominent authorities concerned with this problem.<sup>11-17</sup> Their kind interest and informative replies are most gratefully appreciated. We have been made aware of the fact that the problem is under active consideration by the Subcommittee on Permissible External Dosage of the National Committee on Radiation Protection, headed by Dr. G. Failla. Three hundred milliroentgens, the figure generally accepted for whole body irradiation, is considered by many to be conservative for local exposure, and it is our feeling, based on the replies which we have received, that 600 mr per week to the hands and fingers, or twice the whole body figure, is reasonably safe. This is the interim figure which we have

adopted for our own practice until the official recommendations of the national committee are made known.

*Roentgen Measurement.* Radiation measuring devices employed in these studies were standard and of two types, the ionization chamber and photographic film. An ionization chamber depends upon the ability of roentgen and radium rays to ionize gases. It consists essentially of a chamber for the collection of charged particles which is connected electrically with a counting rate meter. As the radiation beam passes through the chamber, the rays ionize the contained gas in direct proportion to the intensity of the beam, and that degree of ionization is read off a scale calibrated in milliroentgens. The Radiation Survey Meter is an instrument of this type and was used in these studies. Small pen-shaped self-reading dosimeters—ionization chambers—were also used. The other type of monitoring device is dependent upon the reduction of photographic film by ionizing radiation, the degree of blackening produced being related to the type, duration, and intensity of the radiation reaching the film, as well as the film characteristics. One preparation available commercially consists of a dental film packet and metallic filters in a small holder. By means of a pin or clip the film holder may be attached to the clothing. When worn on the chest these film badges give a fairly accurate record of the "whole body" exposure. To estimate the amount of radiation received by the hands and fingers another form of film meter is available. It consists of a plastic ring with a large signet. Within the signet is a small circle of photographic film and several metallic filters. This film ring and other measuring devices used are illustrated in Fig. 1. In Fig. 2 may be seen the manner in which the ring was worn to record radiation exposure of the finger tips. The film rings were obtained from the Atomic Energy Commission, Isotope Division, while the film badges were available commercially. They are also obtainable through the Atomic Energy Commission. At the conclusion of an arbitrary number of exposures the films were returned to their sources for development and comparison with previously calibrated films. Reports were rendered in milliroentgens.

The radium applicators used as sources of radiation for these studies were the Burnam-Crowe type Monel metal nasopharyngeal applicator, wall thickness 0.3 mm, containing 50 mg of radium.

Although quantitative determination of radiation from this applicator can be made with reasonable accuracy, measurements may vary somewhat in the light of certain factors. First, small variations in placement of measuring devices, especially as the radium bearing tube is approached, will cause relatively large variations in readings.

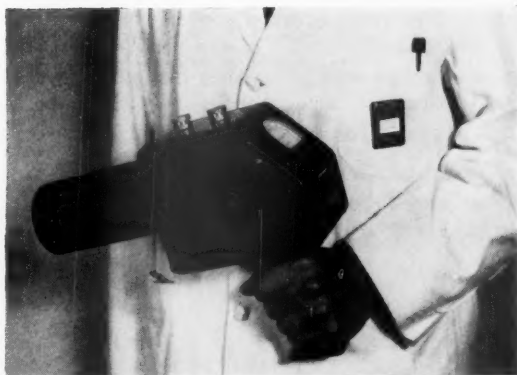


Fig. 1.—Monitoring instruments used in these studies—radiation survey meter, film badge, film ring, pocket electroscope.



Fig. 2.—Film ring mounted for measuring finger tip exposure.

Second, there is often a therapeutically insignificant but quantitative difference in the roentgen output of different applicators. Third, because of so-called backscatter, that property of x-rays and radium rays by which they bounce off metal and other matter, readings may vary from room to room. Lastly variations in the measuring instruments themselves and different arrangements of this equipment in an experiment, especially in cross-checking by different methods, will account for corresponding differences in readings.

*Present Investigation.* Quantitative measurements in milliroentgens\* were made of the roentgen output of the 50-mg nasopharyngeal radium applicator by a number of methods and under a variety of circumstances. All values listed represent the average of multiple measurements.

TABLE 1.  
INTENSITY OF UNPROTECTED APPLICATORS AT VARYING  
DISTANCES.

At surface (Schulz and Robbins <sup>1</sup> )	140 r/min. or 8,400,000 mr/hr.
At surface (Loftus <sup>18</sup> )	150 r/min. or 9,000,000 mr/hr.
Halfway between handling knob and radium capsule	9480 mr/hr.
At the handling knob	1800 mr/hr.
At 1 ft. lateral from the radium bearing capsule	1580 mr/hr.
At 5 ft. lateral from the radium bearing capsule	37 mr/hr.
At 10 ft. lateral from the radium bearing capsule	6 mr/hr.
At 15 ft. lateral from the radium bearing capsule	2 mr/hr.
At 20 ft. lateral from the radium bearing capsule	1 mr/hr.

If these figures are contemplated in the light of official tolerance recommendations of 300 mr per week or 60 mr per working day, it is apparent that lapses in technique will quickly result in overexposure. It is evident that distance affords the greatest protection and that the applicator should never be touched below the handling knob or the radium bearing tube approached from the side.

\*A milliroentgen is one/one thousandth of a roentgen.

TABLE 2.  
MEASURABLE RADIATION THROUGH VARIOUS LEAD CONTAINERS

	ONE APPLICATOR— CLOSED 2 $\frac{3}{4}$ -IN. LEAD CARRYING CASE	ONE APPLICATOR— 4 $\frac{1}{2}$ -IN. LEAD SAFE	TWO APPLICATORS— CLOSED 4 $\frac{1}{4}$ -IN. LEAD CARRYING CASE
At outer surface of case	4500 mr/hr.	500 mr/hr.	1560 mr/hr.
At 1 ft.	90 mr/hr.	24 mr/hr.	60 mr/hr.
At 5 ft.	4 mr/hr.	1 mr/hr.	1 mr/hr.
At 10 ft.	1 mr/hr.	negligible	negligible
At 15 ft.	negligible	—	—
At 20 ft.	—	—	—
At carrying strap	145 mr/hr.		

It is apparent that freedom from exposure does not lie simply in keeping the applicators in lead containers, but that distance is again the most important factor.

TABLE 3.  
PENETRATION OF RADIATION WITH APPLICATOR IN THE  
NASOPHARYNX.

At the handling knob	1600 mr/hr.	
	LATERAL	ANTEROPOSTERIOR
At 1 ft. from center of head	500 mr/hr.	500 mr/hr.
At 5 ft. from center of head	16 mr/hr.	19 mr/hr.
At 10 ft. from center of head	4 $\frac{1}{2}$ mr/hr.	4 $\frac{1}{2}$ mr/hr.
At 15 ft. from center of head	1 $\frac{1}{2}$ mr/hr.	1 mr/hr.
At 20 ft. from center of head	negligible	negligible

Little gamma radiation is absorbed by the patient when the applicator is in position in the nasopharynx. Note that at a distance of 10 ft. continuous exposure in an eight-hour day is well below tolerance for the operator.

*Summation of Hand and Finger Exposure.* In manipulating the applicator the fingers are in the forefront and bear the brunt of exposure. The tips of the first three fingers of the hand holding the applicator and the tips of the index finger and thumb of the hand holding the speculum are consistently closest to the radium bearing tube. A series of 60 treatments was carried out with a signet ring film badge mounted on the end of the right middle finger and 60 with a ring on the tip of the left index finger. These rings were then returned to Oak Ridge for development, and, in due time, a report of the number of roentgens to which each ring had been exposed was received. The figure for each ring was divided by the number of treatments which it had monitored and an average value for each treatment obtained. The right hand finger showed about 50 mr per treatment, the left 60 mr. The average value for the left index finger tip was higher because it was carried far forward on the speculum and subjected to a short-lived but great intensity as the radium bearing tube passed between the blades of the speculum into the nose. In a series of 20 treatments in which the ring was worn lower than the nail bed area on the index finger of the left hand, the average value was 40 mr per treatment. This suggests that during introduction of the applicator, the nasal speculum should be gripped as far from the nose as possible.

To determine the amount of irradiation reaching an entire finger, pen-shaped self-reading dosimeters, ordinarily worn in the vest pocket, were taped to the index finger of each hand and individual readings made during the introductory stages of a series of treatments. Dosage averaged approximately 25 mr for the right index finger and 27 mr for the left. These figures emphasize the protective merit of distance, since the average exposure of a whole finger is but half that of its tip.

Average time for removal of the applicator at the end of a treatment, including loosening the adhesive tape and cleansing the radium bearing tube, is about 30 seconds. If the withdrawal is performed with the right hand, dosimeter readings show that the whole index finger receives a dose of approximately 14 mr. Since a finger tip receives about twice as much irradiation as the entire length of the corresponding finger in this operation, the nail-bed area may be expected to receive about 28 mr during the process of withdrawal. If, as in our technique, the adhesive tape is loosened by a nurse and the applicator then withdrawn by the physician and passed to her for cleansing, the exposure of her index finger tip will be less than 28 mr per treatment. The thumb will of course receive the same amount.

*Total Body Irradiation.* From film badge records and from repeated dosimeter readings, it was shown that the physician receives an average total body dose of 3 to 6 mr and the nurse 4 to 6 mr per treatment. This assumes they remain a distance of 10 ft. from the patient during a 12-min. treatment period. Other personnel show exposure figures far below these. At a patient distance of 20 ft. exposure is only about 1 mr less, an insignificant difference.

TABLE 4.  
SUMMATION OF EXPOSURES TO PHYSICIAN AND ASSISTANT.

POSITION OF FILM OR MONITOR	AVERAGE EXPOSURE PER TREATMENT	PROCEDURE
Right middle finger tip	50 mr	Introduction
Left index finger tip, extended	60 mr	Introduction
Left index finger, 5 cm from tip	40 mr	Introduction
Entire right index finger, average dose	25 mr	Introduction
Entire left index finger, average dose	27 mr	Introduction
Chest (whole body)	3-6 mr	Introduction
Chest (whole body), assistant	4-6 mr	Introduction
Entire index finger, average dose, assistant	<14 mr	Loosening adhesive tape, cleaning applicator

#### DISCUSSION

If the radium applicator is introduced smoothly and with no delay, the time required is 25-30 seconds. This includes anchoring the handle to the cheek with adhesive tape in order to draw the radium bearing tube against the torus tubarius and checking its position with a postnasal mirror. Unfortunately all too many introductions are slowed by adventitious occurrences, and the average time is about 40 seconds. Here are a few common delaying actions which are bound to occur sooner or later in any sizeable series.

1. The otolaryngologist grasps the applicator and poises it for introduction. At this precise moment the patient, either adult or child, shies away to take a deep breath, to ask a question, to sneeze, or to seek further reassurance. Meanwhile the physician futilely holds the applicator until the patient is ready to proceed. Reassurance at this point, with consequent delay, is very often necessary with young children. In anticipation of this apparently universal

occurrence one radium company has recently announced that it includes an identically appearing dummy applicator with each real one for "safe demonstration to the patient and to help break down the psychologic barrier."

2. One nasal passage is frequently narrower than the other and requires that the applicator be insinuated slowly. If, in addition, the narrowing is associated with a low septal deviation, there may be much more than momentary delay as the obstruction is carefully passed. This is most pernicious and is to be particularly avoided since the radium capsule close to the anterior naris is a very short distance from the hand gripping the nasal speculum. Because of the vast rise in intensity output as the radium bearing tube is approached, a delay at this point will subject the fingers of the left hand to a spray of radiation that may at one sitting far exceed permissible dosage for the day.

3. As the tongue is depressed to check the position of the applicator with a postnasal mirror, the patient may gag and inadvertently delay visualization. Omission of this mirror examination in those who will tolerate it is not in accord with best practice because of the disquieting frequency with which an applicator seemingly well placed turns out to be millimeters from the tubal cushion.

4. In those instances where postnasal mirror examination shows that readjustment of the applicator is necessary, the adhesive tape must be repositioned to pull the radium bearing tube more laterally and a mirror check again made. These manipulations draw the fingers onto the face below the level of the handling knob and thus increase the amount of exposure out of proportion to the time.

If a physician simply slips the applicator in and neither anchors it nor checks position with a mirror, naturally his finger exposure will be much less. Since we prefer to draw the applicator sideward to anchor the radium bearing capsule against the tubal cushion, then check where possible with a mirror, we assume, in accordance with our measurements, that the nail bed areas of the first three fingers of the right hand receive an average dose of 50 mr per treatment. Because most children cannot be examined by mirror, this figure might appear high, but some of the above enumerated delays and a further dose incident to withdrawing the applicator, even if it is passed to a nurse for cleansing, will keep exposure at about this 50-mr level. Parts of the thumb and forefinger of the left hand will receive between 40 and 60 mr per treatment, depending upon the manner in which the speculum is held.

If the physician exercises reasonable caution and holds the applicator away from his body, the fingers are the limiting factor in

the number of treatments which may be safely administered by the physician. Since we have adopted a figure of 600 mr per week for finger and hand exposure, or twice the whole body exposure of 300 mr per week, a physician who follows the described technique should limit himself to 12 treatments a week. This is many less than heretofore considered safe.

An assistant, if she helps with no more treatments than are administered by one physician will not, under normal circumstances, receive more adventitious irradiation than the physician. A careless or improperly instructed assistant can, however, very easily exceed tolerance, and the medico-legal implications are clear. In our office we have posted the following detailed instructions on the wall over the radium containers for all personnel handling radium to follow:

1. Never touch the radium applicator anywhere except at the handling knob. Hold the applicator away from the body.
2. Never carry the case other than by the strap provided.
3. When a treatment is to be administered, transfer an applicator quickly from the safe to a small carrying case. Close the case and bring it to the treatment room. Prepare a strip of adhesive tape for anchoring and put it on the treatment table.
4. Never hand the applicator to the doctor. He will withdraw it from the carrying case.
5. Stand back at least 5 ft. as the physician opens the case and introduces the applicator. With children assist in whatever manner is necessary.
6. When the physician has completed the operation, set the timer for 12 min. and escort the patient to a recovery room couch. After making sure the patient is comfortable, leave the room and maintain a distance of at least 10 ft. Look in on the patient occasionally. Allow parents to sit with their children.
7. When the timer bell rings, loosen the adhesive tape in not more than 5 seconds.
8. The physician will remove the applicator and hand it to you.
9. Quickly rinse the applicator under running water and clean with the long handled brush provided for this purpose. Pass the applicator through zephiran solution and replace in case. This must take less than 30 seconds.
10. Return the carrying case to the storage room. Transfer applicators back to the safe only at the end of the day.

11. Keep slots in safe and carrying cases filled with 1:1000 aqueous solution of zephiran.

12. Be quick in your movements and never allow the radium bearing tube to come closer to your fingers or any part of your body than the handling knob.

Otolaryngologic assistants, not having received the indoctrination of x-ray technicians, must be impressed by the physician with need for care and undeviating technique in this work.

It will be noted from the tables that a patient-distance of 10 ft. after placement of the applicator is, for practical purposes, as satisfactory as 20 ft. The output of the radium bearing tube in the patient is only about  $4\frac{1}{2}$  mr an hour at 10 ft., which is equivalent to slightly less than 1 mr in a 12-min. treatment period.

The lead safes furnished as containers call for particular comment, as shown by Table 2, because the protection they afford is to a certain extent illusory. A significant quantity of gamma radiation reaches their outer surfaces, especially the small case. Consequently they should be lifted only by their straps and should be stored at least 10 ft. from areas frequented by personnel. They do not belong in treatment rooms.

#### CONCLUSIONS

1. In a carefully monitored series of treatments with the 50-mg Monel metal nasopharyngeal radium applicator total body irradiation did not exceed 6 mr per treatment for the physician and assistant. The tolerance dose for whole body is 300 mr per week.

2. Average exposure of the entire length of both index fingers of the physician was 26 mr and of several finger tips 50 mr per treatment.

3. Pending official recommendation we have adopted a figure of 600 mr per week as permissible exposure for hands and fingers.

4. In accordance with this value the maximum number of treatments which one physician may administer weekly by our technique is 12, finger tip and not whole body exposure being the limiting factor.

5. Our results suggest that generally accepted recommendations as to the number of treatments physicians may administer without endangering themselves are too high for safety.

6. Lead shields or other accessories for the radium applicator or modifications in its construction might reduce the dose delivered

to the physician's finger tips and thereby permit a larger number of treatments.

7. If the technique of any physician differs appreciably from the one herein described, it should be monitored to ascertain the number of treatments he may administer by his method without exceeding tolerance.

We gratefully acknowledge the kind co-operation and advice of Doctor Henry L. Jaffe, Director of the Department of Radiation Therapy, Cedars of Lebanon Hospital.

3875 WILSHIRE BLVD.

#### REFERENCES

1. Schulz, M. D., and Robbins, L. L.: Dangers of Irradiation of Hypertrophied Lymphoid Tissue of the Nasopharynx, *Tr. Amer. Acad. Ophth. and Otolaryng.* (Jan.-Feb.) 1949, p. 243.
2. Morgan, K. Z.: Protection against Radiation Hazards and Maximum Allowable Exposure Values, *J. of Ind. Hyg. and Toxicol.* 30:286 (Sept.) 1948.
3. Taylor, L. S.: X-ray Protection, National Bureau of Standards Handbook, No. 15, 1931.
4. Taylor, L. S.: X-ray Protection, National Bureau of Standards Handbook, No. 20, 1936.
5. A Combined Report of the Standardization Committees of the American Roentgen Ray Society and the Radiological Society of North America: Protection against X-rays and Gamma Rays, *Radiology* 46:57 (Jan.) 1946.
6. Henshaw, P. S.: Experimental Roentgen Injury: IV. Effects of Repeated Small Doses of X-rays on Blood Picture, Tissue Morphology, and Life Span in Mice, *J. Nat. Cancer Inst.* 4:513 (Apr.) 1944.
7. Lorenz, E., Heston, W. E., and Eschenbrenner, A. B.: Biological Studies in the Tolerance Range, *Radiology* 49:274 (Sept.) 1947.
8. Sievert, R. M.: The Tolerance Dose and the Prevention of Injury Caused by Ionizing Radiation, *Br. J. Radiol.* 20:306 (Aug.) 1947.
9. Prosser, C. L.: The Clinical Sequence of Physiological Effects of Ionizing Radiation in Animals, *Radiology* 49:299 (Sept.) 1947.
10. Medical X-ray Protection up to Two Million Volts: Handbook 41, Natl. Bureau of Standards, Washington, D. C., 1949.
11. Personal Communication from Doctor G. W. Morgan, Assistant Chief, Advisory Field Service Branch, Isotopes Division, Oak Ridge Operations.
12. Personal Communication from Doctor Fred A. Bryan, Assistant Project Director, Atom Energy Project, University of California at Los Angeles.
13. Personal Communication from Doctor G. Failla, Professor of Radiology, College of Physicians and Surgeons, Columbia University, and Chairman of the Subcommittee on External Irradiation of the National Committee for Radiation Protection.
14. Personal Communication from Doctor Roger A. Harvey, Professor of Radiology and Head of Department, University of Illinois College of Medicine.
15. Personal Communication from Doctor H. M. Parker, Manager, Health Instruments Divisions, Nucleonics Department, General Electric Company.
16. Personal Communication from Doctor Edith H. Quimby, Associate Professor of Radiology, College of Physicians and Surgeons, Columbia University.
17. Personal Communication from Doctor Robert S. Stone, Professor of Radiology, University of California Medical School.
18. Personal Communication from Mr. G. H. Loftus, Vice-President, Radium Chemical Company.

## VII

### EVALUATION OF IRRADIATION OF PHARYNGEAL AND NASOPHARYNGEAL LYMPHOID TISSUE

FRANCIS L. LEDERER, M.D.

CHICAGO, ILL.

Impelled by reports of enthusiastic acceptance, as contrasted by dire warnings of the dangers of irradiation of the nasopharyngeal lymphoid tissue, we wish to offer our own concepts of the problem. There is historical precedence for this attempt because some 27 years ago I recorded my experience on this very subject.<sup>1</sup> Then, as now, there was a mass hysteria which threatened to upset the rational scientific thinking of medical men, not ordinarily given to falling for ballyhoo. Were I to take my report of 1922 and change the date to 1949, modern concepts of the problem would be represented.

The irradiation of lymphoid tissue for deafness is not new. I can recall that some 25 or more years ago at a meeting of this society, Richardson of Washington, D. C., was quite enthusiastic about this mode of therapy for deafness, and that in 1923, Frey and Kriser<sup>2</sup> described its use in the treatment of otosclerosis.

In 1926, Campbell Smyth,<sup>3</sup> at the request of H. P. Mosher, undertook the study of 100 cases of deafness treated with x-ray. He chose such cases where redundant lymphoid tissue was present in the nasopharynx, especially in the lateral pharyngeal wall near the entrance of the eustachian tube. Treatment was given every ten days for six exposures, through two portals directed to the upper pharynx and eustachian tubes. The factors used were 8-in. spark gap, 5 milliamperes, 3 mm aluminum filtration, 12-in. distance, from 5 to 8 minutes. Audiometric control was used before and after treatment. Smyth concluded that deafness and tinnitus of short duration and recurrent tubal deafness were definitely improved. The most suitable group were young children between 8 and 15 years of age. He noted that there was no relation between improvement and the macroscopic appearance of the pharynx.

---

From the Otolaryngological Services of the Illinois Eye and Ear Infirmary, Department of Public Welfare and the University of Illinois College of Medicine, Chicago, Illinois.

Presented before the meeting of the Chicago Laryngological and Otological Society, Chicago, Illinois, March 7, 1949.

Also in the same year, there appeared a report on the treatment of middle ear deafness by means of radium by Stevenson and Wilson.<sup>4</sup> They used emanations, utilizing gamma rays in glass capillary tubes placed in gentle contact with the tympanic membrane. Only eight cases were reported but the authors felt there was promise in this form of treatment of nonsuppurative middle ear disease.

We should always understand why such modalities take hold of the imagination, if not the fantasy, both of the lay public and the medical groups. Firstly, they are motivated by a desire to do something for conditions for which they otherwise can seemingly offer nothing. Secondly, conservatism, even though it may imply inadequacy, has an appeal, especially when the instrumentation appears technically simple and is apparently without danger. Thirdly, a non-surgical approach will have its adherents among those unprepared to assume full surgical responsibility and especially to the layman, to whom abhorrence of anything surgical comes quite naturally. Last, but not least, comes the press, both lay and scientific, heralding the panacea-like results of irradiation, influencing the employment and even coloring the interpretation of results from such a form of treatment.<sup>5-23</sup>

Paradoxically, there were, among these, innumerable contributions to the literature dealing with the unpleasant end-results in well performed tonsil and adenoid operations and also those discussing unsuccessful operations. A deluge of articles followed, advocating irradiation of pharyngeal lymphoid tissue. Such authors went so far as to show the impossibility of removing all of Waldeyer's ring by surgical means, as though this were a necessity. This literary tirade was a reminder that epochs repeat themselves, for in 1912 MacKenzie,<sup>24</sup> writing on the "Massacre of the Tonsil" remarked that, "Never before in the history of medicine has the lust for operation on the tonsils been as passionate as it is at the present day," when, "the minds of the younger generation of operators have been poisoned by incontinent talk" as to the diseases induced by these organs. He also added that removal of the tonsil "is a capital operation, a dangerous operation," which has to its credit, "a long roll of unrecorded deaths." "It occasionally happens," he caustically remarked, "that the resurrection of the 'buried' tonsil is followed by the burial of the patient."

Furthermore, as part of the proof of the desirability for irradiation, they pointed out that many of their patients were children of physicians! From 1922 to 1924, enthusiastic reports, mainly from nonotolaryngological sources, flooded the literature and then, sud-

denly, something happened—no more was said and the method was apparently relegated to oblivion.

My discussion in 1922 was the only negative appraisal to appear. At that time, as now, I acknowledged the possible shrinking effect but emphasized the inability of this form of treatment to function as a bactericidal agent or to control, indefinitely, the hyperplastic tendencies of lymphoid tissue. Adverse effects of extreme dryness in the pharynx was also described and caution in the use of so potent a form of treatment was mentioned. I have continued to use irradiation over 27 years and have yet to observe any neoplastic change occurring in any of the patients treated. Not all of my patients have been treated for deafness. I have taken such patients in age groups not eligible for surgery, by my criteria, or who seemed poor risks at the time, and subjected them to irradiation. This was contemplated merely as a palliative measure to reduce the mechanical obstruction, knowing, of course, that the tissue would not respond permanently. Personal communications with radiologists using both x and r irradiation over three or four decades fail to reveal a single case of neoplastic change in the nasopharynx. B. H. Orndoff<sup>39</sup> believes that an extensive survey would fail to find any sizeable group where radiation damage has occurred in children from this form of treatment. To him, it seems certain that considerably greater doses are well tolerated but unnecessary. On the other hand, cases do exist where other types of lesions in this region, such as nevi, have been treated and have shown damaging results.

In 1939, Crowe and Baylor<sup>25</sup> started a revival of this form of therapy, which, in the original communication, was directed at the prevention of deafness. What diverse ramifications of the original indications have taken place in the past ten years is attested to by the literary inundation which has followed in its wake. In this transcendency several technical changes and alterations in dosimetry have been suggested (I remember the furore created at the American Laryngological, Rhinological and Otological Society meeting in Saint Louis in 1947 when Dr. Goodyear suggested upping the dosage from 8½ minutes!) and a wider application of the use of radium and/or its emanations have been heralded by otologists and non-specialistic groups alike. With startling impact, the Editorial<sup>26</sup> of September 18, 1949, in the *Journal of the American Medical Association*, assailed such indiscriminate employment of so pernicious an agent as radium to the nasopharynx. To add further to this warning, Schulz and Robbins<sup>27</sup> stressed the late effects from irradiation. While they called attention to the possible untoward effects so well known to all clinicians, they failed in what they intended to be

comparable or analogous situations. Much of what they demonstrated was the result of accidental exposure to the face or other parts of the body that had nothing to do directly with the nasopharynx.

When Cutler, Marcus and I<sup>28</sup> presented a report of our experiences in a series of carefully selected and controlled cases of deafness, we expressed the view that irradiation has been effective when combined with other forms of therapy. This was especially true in the child with a syndrome of impaired hearing of a transitory character and allergic symptoms; i.e., difficult nasal breathing, postnasal discharge, sneezing, coughing, otalgia and mouth breathing. Associated paranasal sinus infections were invariably present. We were certain, just as others were, that young lymphoid tissue responded but not necessarily with permanence, and that adult lymphoid tissue showed greater resistance to irradiation. The effects noted in those cases with apparent irreversible hearing loss, such as otosclerosis, adhesive process and nerve deafness were disappointing, but not unexpectedly so. The mild tubal disturbances, especially in the young, gave the more favorable results such as have been experienced by Crowe,<sup>29</sup> Scal,<sup>30</sup> Boies,<sup>31</sup> Fowler,<sup>32</sup> Fricke and Brown,<sup>33</sup> Harris and Montgomery,<sup>34</sup> and Gay.<sup>35</sup>

Crowe stressed the effects of irradiation on the immediate vicinity of the pharyngeal orifice of the eustachian tube, with probable beneficial effects on the lymphoid tissue throughout the pharynx. Radiologists studying the factors of a 0.3-mm Monel metal applicator, using either 50 mg of radium salt or 50 millicuries of radon as either an immediate postadenoidectomy procedure or in cases of so-called granular lymphoid hyperplasia following adenoidectomy, are not agreed that Crowe is correct in assuming a harmlessness of application. Even using the three 8½-min. applications, at 2-week intervals, it seems that each application delivers over one minimal skin erythema dose to the mucosa of the lateral wall of the pharynx. While it may be assumed that the vascularity of the pharyngeal mucosa renders it more tolerant to irradiation than the average skin surface, it is only fair to point out that even the 8½-min. dosimetry of three treatments would produce skin changes in the average patient.

T. J. Wachowski,\* a radiologist of considerable experience and from whom I have obtained most of my information, challenges Crowe's statement that such a series can be repeated in 30 to 90 days (apparently without producing damage). Advocacy of this

\*Professor of Radiology, University of Illinois College of Medicine.

idea would not be accepted by the average radiotherapist. While Crowe's cases have not shown such ill effects, it cannot be assumed that misuse of the pharyngeal applicator in the hands of the average otologist will be so innocuous. This is especially true when deafness from any cause and without a proper inspection of the nasopharynx is going to be treated. In many of these cases, not only is the treatment not indicated but lack of response after the first try is frequently followed by a second course of irradiation. Dr. Wachowski tells me that he has heard of cases where the patient was referred for roentgen therapy for deafness after having received up to three series of pharyngeal radium applications!

The history of previous irradiation is of extreme importance. This question will now rank equally with the family history of deafness. While no malignant transformation resulting from this type of therapy has been reported, the possibility of malignancy from repeated courses of therapy by succeeding therapists must be considered. Recent reports on the incidence of carcinoma of the body of the uterus following the use of radium for benign uterine conditions, places its occurrence at 8%, far beyond the normal. While the conclusion is still controversial, and unproven in regard to the nasopharynx, numerous investigators have found that intensities of radiation weaker than injurious quantities may stimulate cells and tissues to increased metabolism. Lobe<sup>36</sup> concludes, "as a result of often repeated stimulation of tissues which are able to respond with growth processes, at last a transformation may be accomplished in which the effects of stimulation are not temporary, but in which the effects have become permanent; or expressed differently, in which cancer has been produced." Cade<sup>37</sup> points out that it is the continuous exposure over a prolonged period which constitutes the danger to the radium worker as distinguished from the relatively short exposure of the patient. Attention to the history of previous therapy particularly with x and r irradiation should prevent any danger of overexposure. It is of passing interest also to note that two clinicians have developed skin changes on their hands.

In the evaluation of this therapeutic application, we must call attention to the fact that, depending on the source of the beta ray, the procedure differs. While x and r irradiations include both beta and gamma rays, an equation cannot be derived except in the vague term of biologic effect. The output of beta ray from radium sulfate and from radon differs, despite the fact that their gamma ray emanation has been standardized. The millicurie of radon is that quantity which exhibits the same gamma ray activity as a milligram of radium. Crowe<sup>38</sup> calls attention to the assay of Bernard T. Feld

which showed that the 0.3-mm Monel metal applicator containing radon, had a beta ray output two and one-half times that of a similar applicator containing radium sulfate. It would seem that more stress should be placed upon the criteria for the use of the pharyngeal beta ray applicator, and that more investigative work be done on the biological dose given off by the beta rays in the nasopharynx, correlated with the present standard, viz., the minimum skin erythema dose, suggested by Schulz and Robbins.<sup>27</sup>

I feel that Wachowski's remarks are so pertinent and represent the opinions of those radiologists with whom I have had professional contact these many years, that I am recording them in detail:

"Personally, I have used the pharyngeal radium applicator only infrequently, although I had one at my disposal for two years. I found that few cases sent to me met Dr. Crowe's criteria for the use of the applicator. Most of the cases I saw were children under six, in whom the attending physician did not care to do a tonsillectomy and adenoidectomy because of the frequency of regrowth of the lymphoid tissue, and was trying to avoid the double expense of surgery plus irradiation; children, and some adults, in whom, to me, the major pathology was in the paranasal sinuses and who exhibited periodic pharyngitis during exacerbation of their sinusitis.

"Since Crowe's work would seem to discredit the presence of the so-called 'tubal catarrh' as an entity dissociated from lymphoid obstruction of the ostium, and since many of the cases I saw did not have proper nasopharyngoscopic investigation, I hesitate to mention this condition. I believe it is true, however, that the lymphoid tissue can extend into the eustachian tube. Since the zone of influence of the Crowe applicator is very limited, practically all of the beta rays being absorbed in the first 5-7 mm of tissue, such cases would not be benefited by beta therapy. I felt that roentgen therapy, using high voltage with filtration sufficient to give a half value layer of 1 mm of copper, was indicated in most cases that I saw. This opinion was based on the diffuse disease seen in most cases and on the fact that the pharyngeal infection subsided with small doses of roentgen rays. In most cases, the lymphoid hyperplasia also subsided gradually to a satisfactory level, over a period of one to three months.

"Where the infectious manifestations were most prominent, I used what I shall call *Technique 1*, delivering 100 r (in air) to portals covering the entire sinus and pharyngeal areas, treating one side at a time. Sides were alternated and treatments were given every three or four days until each side had received 100 r, times two.

Where the hyperplastic lymphoid manifestations were most prominent, I gave what I shall call *Technique 2*, delivering 100 r (in air) to both sides of the face and pharynx at one sitting. This dose was repeated four times at weekly intervals (400 r, times four to each side). Occasionally, if the films of the paranasal sinuses showed no involvement and symptoms and findings of sinus disease were absent, only the pharynx was treated (100 r, times four to each side). In very young infants, the dose was reduced to 50 r, times four, and in children of three to four years of age, the average dose was 75 r, times four.

"Technique 2, usually delivered 100 to 125 r per sitting to the midline of the pharynx in the average child (facial diameter 12-14 cm at the zygoma), and Technique 1 gave one-half of that dose per sitting.

"My satisfactory experience, based on clinical follow-ups by a pediatrician and otorhinolaryngologist, is similar to that which other radiologists have been having for many years. It is only logical that the results of such therapy should be satisfactory, because of the sensitivity of the lymphoid elements to radiation, particularly in the young. It is my impression that much of the lymphoid enlargement is conditioned by chronic infection. Subsidence of the infection is followed by regression of the lymphoid mass.

"It has been my experience that results have been better since I have been using the larger portals. I believe it is impossible to dissociate the mucous membrane of the pharynx, sinuses, eustachian tube, and middle ear from one another. Proper localization of the beam makes me feel certain that the pituitary gland (mentioned by Dr. Crowe in a discussion of roentgen therapy) is not receiving any significant radiation. Children can easily be mummified for immobilization. Since 550-600 r constitute a minimum skin erythema dose under the conditions I use, it is obvious that the roentgen therapy gives a smaller, more diffuse exposure than the Crowe radium applicator.

"In résumé, I have no objection to the employment of the Crowe beta ray pharyngeal applicator when used after adenoidec-tomy or for small, recurrent lymphoid masses, and in reasonable doses. I feel that repeated exposures with the Crowe applicator are not so innocuous as some of his writings lead us to believe. For most of the pharyngeal conditions sent to me for radiation therapy, I feel that high voltage, properly filtered and properly localized roentgen rays are preferable."

The histological changes of the faucial and pharyngeal tonsils following irradiation have been described by others and have been confirmed by us. Three days after irradiation there is a swelling of the protoplasm with degenerative changes of the nuclei in the center of the follicle. About three weeks later, one can no longer observe the difference between the nucleus and the cytoplasm. There is an exudation and a cellular infiltration in the irradiated area of the tonsil. The typical lymph-adenoid tissue, i.e., the follicular formation, disappears completely and is replaced by an indifferent form of granulation tissue. After six months, the tonsillar tissue consists mainly of connective tissue. This new connective tissue, in contrast to the old connective tissue, lacks elastic fibers. It is of interest to note that the epithelial layer evidences only slight changes, such as the lack of mitosis of the basal layer, but otherwise few inflammatory cell changes occur. Still later, in some cases, cartilaginous replacement of the connective tissue may take place and here and there small ossifying areas are occasionally noted.

#### SUMMARY

1. Considering all of the available evidence, irradiation of the nasopharynx by the standardized techniques is without danger. It should, however, be used with restraint and in technically knowledgeable hands, because of our incomplete knowledge of radiobiology and the empiricism of the present dosage.

2. Its use is not a substitute for surgery and should be confined to those cases of conduction deafness associated with the proven presence of lymphoid hyperplasia in and about the eustachian tube orifices.

3. The syndrome of transitory hearing loss, allergic manifestations and infection in the nose, sinuses and/or nasopharynx must be recognized and treated accordingly, in addition to irradiation.

4. The importance of history-taking in relation to previous irradiation is stressed because of the possibility that a repeated series of treatments may produce untoward tissue changes.

5. The warning of the danger to the careless technician himself must be respected.

6. The use of irradiation in irreversible hearing deficits is to be deplored and discouraged.

7. We are agreed that in competent hands, no great advantage is to be had from r over x irradiation. As a matter of fact, the simplicity of obtaining and carrying out the first form of therapy may eventually lead to the harm which we wish to avoid.

8. The increasing prevalence of the routine postoperative irradiation of the nasopharynx is purely an admission on the part of the otolaryngologist that this operation is technically inadequate.

9. Histologically, all stages of degeneration in the lymphoid tissue may be demonstrated following irradiation. This is then followed by varying degrees of reparative change from granulation tissue, to localized areas of ossification in occasional cases. No further metaplastic change has been noted nor has there been any alteration of the surface epithelium.

1853 WEST POLK STREET.

#### REFERENCES

1. Lederer, Francis L.: The Roentgen Ray in Tonsillar Disease, J. A. M. A. 69:1130, 1922.
2. Frey, H., and Kriser, A.: Therapeutic Studies with Roentgen Management in Otosclerosis, Zeitschrift f. Hals, Nasen und Ohrenh. 6:334-338, 1923.
3. Smyth, D. Campbell: Treatment of Lateral Pharyngeal Bands by X-rays in Cases of Deafness, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 35:1157 (Dec.) 1926.
4. Stevenson, W. C., and Wilson, T. G.: Treatment of Middle Ear Deafness by Radium; Its Rationale and Technique; Preliminary Report, J. Laryng. and Otol. 42:96-104 (Feb.) 1927.
5. Witherbee, W. D.: Contraindications and Results in Surgical Removal of Tonsils and Adenoids Compared with X-ray Therapy, Am. J. Electrotherap. and Radiol. 11:375-377, 1922.
6. Babcock, J. W.: Observation on the Results of Roentgen Therapy in Chronic Tonsillitis, J. A. M. A. 70:300, 1923.
7. Lane, Laura A.: A Study of the Tonsil Question with a Preliminary Report of Roentgen Ray and Radium Therapy in the Treatment of Pathologic Tonsils, Minn. Med. 6:97-104, 1923.
8. Nogier, T.: Treatment of Hypertrophied Tonsils with Roentgen Therapy, Paris Med. 48:109, 1923.
9. Robinson, C. F.: The Present Status of Radiation Treatment of Tonsils, New York M. J. 117:39, 1923.
10. Robinson, C. F.: Radium Treatment of Diseased Tonsils, Am. J. Roentgenol. 9:588-591, 1922.
11. Williams, F. H.: Treatment of Hypertrophied Tonsils with Radium Therapy, Paris Med. 47:110-113, 1923.
12. Williams, F. H.: The Treatment of Tonsils by Radiations from Radium Salts Instead of Operations; 101 Cases, Tr. Assn. Amer. Phys. 37:204-212, 1922, also Boston M. and S. J. 188:412-416, 1922.
13. Williams, F. H.: The Use of Radium Radiations in the Treatment of Tonsils; A Further Report, Am. J. M. Sc. 168:1934, 1924.
14. Wallerstein, E. U.: Treatment of the Infected Tonsils and Adenoids, Virginia M. Month. 50:177-180, 1923-24.
15. Blaine, E. P.: X-ray Therapy of the Infected Hypertrophied Tonsil, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 33:534-540, 1924.
16. Sewell, E. C.: Roentgen-ray Treatment of the Tonsils, Progr. Med. 1:265-269, 1924.

17. Lewis, E. R.: Fundamental Considerations Underlying Roentgen-ray Therapy of Tonsils, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 33:198-204, 1924.
18. Hunter, J. W., Jr.: The Radiological Treatment of Diseased Tonsils, *Virginia M. Month.* 2:12-15, 1924-25.
19. Denman, I. O.: Triad of Radiation in Pharyngo-tonsillar Infections, *Am. J. Phys. Therap.* 1:121-125, 1924.
20. Denman, I. O.: The Effect of Radiation upon Tonsils and Adjacent Structures, *J. Am. Assn. Med. Phys. Research.* 2:77-79, 1924-25.
21. Simpson, C. A.: Radium Treatment of Tonsils, *Am. J. Roentgenol.* 12: 527-530 (Dec.) 1924.
22. Watkins, W. W.: Pathologic Basis for Roentgen-ray Treatment of Tonsil Disease, *J. A. M. A.* 83:1305-1308, 1924.
23. Scal, J. C.: Diseased Tonsils Treated with Radium, *Am. J. Phys. Therap.* 2:163-165, 1925-26.
24. MacKenzie, John R.: The Massacre of the Tonsil, *Maryland M. J.*, September, 1912; Editorial Comment, *J. Laryng. and Otol.* 27:46 (Dec.) 1912.
25. Crowe, S. J., and Baylor, J. W.: Prevention of Deafness, *J. A. M. A.* 112:585 (Feb. 18) 1939.
26. Editorial, The Hazards of X-ray, *J. A. M. A.* 138:214-215 (Sept. 18) 1949.
27. Schulz, M. D., and Robbins, L. L.: Dangers of Radiation Therapy of Hypertrophied Lymphoid Tissue of the Nasopharynx, *Trans. Am. Acad. Ophth. and Otolaryng.* 243:253 (Jan.-Feb.) 1949.
28. Cutler, M. H., Marcus, R. E., and Lederer, F. L.: American College of Radiology, Refresher Course, March, 1948, Chicago. *Radiology* 52:816-818 (June) 1949.
29. Crowe, S. J.: Irradiation of Nasopharynx, Wherry Memorial Lecture, 51st Annual Session of the Am. Acad. Ophth. and Otolaryng., Oct. 13-18, 1946, Chicago, Ill.
30. Scal, J. C.: Radiation Therapy for Prevention of Deafness and Treatment of Upper Respiratory Infections, *Eye, Ear, Nose and Throat Month.* 27:64-71 (Feb.) 1948.
31. Boies, L. R.: Irradiation of Pharyngeal Lymphoid Tissue, *Arch. Otolaryng.* 44:141 (Feb.) 1944.
32. Fowler, E. P., Jr.: Irradiation of the Eustachian Tubes, *Arch. Otolaryng.* 43:1 (Jan.) 1946.
33. Fricke, R. E., and Brown, H. A.: Radium Treatment of Nasopharyngeal Lymphoid Hypertrophy, *South. M. J.* 37:399-402 (July) 1944.
34. Harris, H. E., and Montgomery, E. L.: Treatment of Lymphoid Hyperplasia of Nasopharynx by Radium, *Cleveland Clin. Quart.* 13:117-124 (July) 1946.
35. Gay, L. N.: Treatment of Residual Lymphoid Tissue in Nasopharynx by Radium, *J. Allergy* 17:348-351 (Nov.) 1946.
36. Lobe, L.: Effects of Radiant Energy, *J. Cancer Res.* 7:229-282, 1922.
37. Cade, Sir A.: Malignant Disease, Baltimore, The Williams and Wilkins Company, Ed. 2, Vol. 1, 1948.
38. Crowe, S. J.: Change in Technique for the Monel Metal Radium Applicator Used in the Treatment of Hyperplastic Lymphoid Tissue in the Nasopharynx, New York, Radium Chemical Company, Inc., 1947.
39. Orndoff, B. H.: Personal communication.

## VIII

### SURGICAL TREATMENT OF BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

JOHN H. BARRETT, M.D.

HOUSTON, TEXAS

Until the past decade the patient who had the misfortune of having a bilateral abductor paralysis of the vocal cords could be offered very poor rehabilitation of the larynx. The choice was between invalidism and permanent tracheotomy. Although a limited group of patients could tolerate a bilateral abductor paralysis, the majority sought surgical relief. A Tucker<sup>24</sup> valvular tracheotomy tube enabled the individual to speak without plugging the tracheotomy opening but the metal tube caused tracheal irritation and was a source of embarrassment, especially to women patients. Nerve repair was advocated by Ballance,<sup>1</sup> Frazier,<sup>4, 5</sup> Lahey<sup>13</sup> and others<sup>2, 6, 14</sup> but the results were unsuccessful due to the dual function of the recurrent laryngeal nerve. Cordotomy<sup>9</sup> and cordectomy<sup>8, 16</sup> were heroic procedures aimed at removing one paralyzed cord to secure an adequate glottic space. After these procedures the voice was very poor and scar tissue formation produced symptoms as distressing as the bilateral paralyzed cords.

In 1929 King<sup>12</sup> published his preliminary report of an operation to restore function to one of the paralyzed cords. His operation was based upon orthopedic procedures employed in the treatment of paralysis resulting from poliomyelitis in which a viable muscle is transplanted to replace a functionless one. Through an incision along the anterior border of the sternomastoid muscle the arytenoid cartilage was exposed and the severed anterior belly of the omohyoid muscle was attached to the muscular process. In cases of long duration the ankylosed crico-arytenoid joint was mobilized by dividing the attachment of the interarytenoideus muscle and severing the joint capsule. The arytenoid cartilage was displaced outward and anchored to the thyroid cartilage until the attachment of the omohyoid

---

From the Department of Otolaryngology, Baylor University College of Medicine, Houston, Texas.

This is part of material presented as a candidate's thesis to the American Laryngological, Rhinological and Otolological Society.

Read before the Texas Society of Ophthalmology and Otolaryngology, December 2, 1949, San Antonio, Texas.

was firm. Later it was realized that the success in this operation was the result of immobilization of the arytenoid and fixing it in abduction rather than restoration of function to one of the vocal cords. Of the many<sup>17-19, 21, 22</sup> modifications of the King operation, Clerf's procedure<sup>4</sup> is one of the most applicable. In 1941, when it was still thought that the King procedure was a function restoring operation, Kelly<sup>10</sup> reported a procedure for extralaryngeal removal of the arytenoid cartilage through a window in the lamina of the thyroid cartilage. He felt that the good results in the King operation were due to the outward displacement of the arytenoid cartilage and that by completely removing the cartilage the cord could be more successfully fixed in a position of abduction. In addition the space created by removal of the arytenoid would further insure adequate glottic space. Of the many<sup>7,20,26</sup> modifications of the Kelly operation the procedures advocated by Woodman<sup>25</sup> and Thornell<sup>23</sup> are of special interest. Woodman employs an incision along the anterior border of the sternomastoid and exposes the arytenoid by disarticulating the inferior cornu of the thyroid cartilage from the cricoid. After removal of the arytenoid the suture through the vocal muscle is passed around the inferior cornu and sutured to the sternomastoid muscle. Thornell<sup>23</sup> exposes the larynx with a Lynch suspension apparatus and removes the arytenoid through an intralaryngeal approach. This procedure requires that the operator be adept in the use of the suspension laryngoscope.

*Comparison of the King and Kelly Procedures.* Today the unfortunate patient with a bilateral abductor paralysis can expect to be relieved of his dyspnea and still retain a serviceable voice. The surgeon's problem is a decision as to which procedure he considers superior in his hands. Reference<sup>15</sup> has been made to the operation as the Kelly-King procedure. This is not an applicable term; though both are cord displacement operations the desired results are accomplished by different procedures. Time and observation have proven that the King operation is not a function restoring procedure and transposition of the omohyoid muscles is no longer employed. The success of the procedure depends upon anchoring the arytenoid cartilage in an outward position after the cartilage has been mobilized by division of the joint capsule and the attachment of the interarytenoid and posterior crico-arytenoid muscles. The success of the Kelly operation depends upon complete removal of the arytenoid cartilage and fixation of the vocal cord in a position of abduction. They are both extralaryngeal procedures which avoid scar tissue formation in the interior of the larynx. There are advantages and disadvantages to each operation. In the King procedure the approach is easier and there is less chance of injury to the mucosa of

the larynx. There are several disadvantages to this procedure. It entails considerable tedious dissection in severing the muscle attachments and joint capsule from the arytenoid cartilage. The splinting suture must be passed through or around the arytenoid cartilage and is likely to fracture it, causing the cartilage to act as a foreign body or allowing the anchoring stitch to pull through. Any disproportion in the size of the arytenoid cartilage and spread of the wings of the thyroid cartilage may make it difficult or limit the degree to which the arytenoid can be rotated laterally. Faulty execution of any one of these steps could mean failure of the entire operation. The Kelly operation offers two distinct advantages. Removal of the arytenoid cartilage frees the paralyzed cord from all structures which fix it in the midline, allowing it to be sutured in a position of abduction. The space left by removal of the arytenoid cartilage is obliterated by scar tissue contraction and increases the size of the glottis. Kelly<sup>11</sup> has referred to some of the difficulties encountered in his procedure. There may be troublesome hemorrhage, especially from a small branch of the superior thyroid artery which may be injured in the upper part of the window. The arytenoid may be freely movable making it difficult to remove and the laryngeal mucosa may be lacerated in dissecting out the cartilage. There may be difficulty in exposure, especially in patients with short, thick necks or in those who have had secondary thyroidectomies or x-ray treatment. Morrison<sup>19</sup> points out the difficulty most surgeons have in operating through the small opening, three-eighths inch square, in the lamina of the thyroid cartilage. Neither procedure pays much attention to postoperative cosmetic results as both leave the patient with two or more scars on the neck. In either procedure there may be infection with subsequent perichondritis or the mucosa of the larynx or pharynx may be injured. Operative trauma and scar tissue formation may cause difficulty in exposing the larynx regardless of the procedure employed. Woodman's approach<sup>25</sup> has one distinct advantage in incising along the posterior border of the wing of the thyroid cartilage and disarticulating the inferior cornu. Both of these landmarks can be identified in any neck regardless of distortion of the normal anatomy. The King operation seems to be more popular among the general surgeons while the Kelly procedure is more applicable to the otolaryngologist who is already experienced in subperichondral dissections.

*Observations.* In the selection of the more suitable procedure the author was prejudiced by having been trained under Joseph D. Kelly. However, this personal contact revealed the difficulties that could be encountered in this operation. The King operation was considered but was not selected as the preferable procedure. After



Fig. 1, Case 1.—Photograph showing three scars on neck after thyroidectomy, tracheotomy and arytenoidectomy with incision along the anterior border of the sternomastoid muscle.



Fig. 2.—Photograph showing position of tracheotomy in old thyroid scar after arytenoidectomy had been performed.

performing the operations both on cadavers in the anatomy laboratory and fresh specimens in the morgue, removal of the cartilage was deemed a more satisfactory procedure than mobilization of the arytenoid and passing a suture through or around it. However, the approach through a half Kocher incision and a window in the lamina of the thyroid cartilage did not afford adequate exposure. In the first case operated upon Woodman's modification of the Kelly operation was employed and the cord was transfixed and the suture passed around the inferior cornu to the sternomastoid muscle as Woodman advocated. The results were satisfactory in that the patient could be decannulated, and that she had a serviceable voice. The operated cord was displaced downward and laterally rather than directly lateral. This patient's voice was not as good as Kelly obtained by his window technique. This patient had three disfiguring scars on her neck which, in her case, caused no concern (Fig. 1). In another case the operation was performed through a half Kocher incision, the exposure was inadequate and the laryngeal mucosa was perforated, resulting in a postoperative infection. The next patient, a young woman who had been under treatment for a depressive psychosis, was distressed about the poor voice and the disfiguring scars which the operation would cause. In this case an incision was made through the old thyroidectomy scar, similar to the incision employed by Shirer,<sup>22</sup> and the skin flap was elevated until the posterior border of the thyroid cartilage and the inferior cornu could be dissected free. After removal of the arytenoid cartilage the anchoring suture in the thyro-arytenoid muscle was passed through the lamina of the thyroid cartilage by means of a straight Lane cleft palate needle. This procedure brought the anchoring suture through the wing of the thyroid cartilage in the same location as the window employed by Kelly. The resulting voice was much better and the patient did not have an additional scar on her neck. In subsequent operations this incision and the technique of passing the fixation suture through the lamina of the thyroid cartilage has been employed with satisfactory results. If a preliminary tracheotomy has not been performed it is made in the thyroidectomy scar, and the patient ends with only one scar (Fig. 2). All cases of bilateral abductor paralysis in which an arytenoidectomy was performed followed thyroidectomy; three others have been seen, two due to poliomyelitis and the other to intracranial metastasis. The paralysis had been present in all cases for at least six months. In removing the arytenoid the perichondrium and muscles are elevated and dissected free with a Freer knife to avoid injuring the laryngeal mucosa. Usually the entire cartilage, as well as the corniculate cartilage, can be removed. In two cases the muscular process fractured off and was not re-

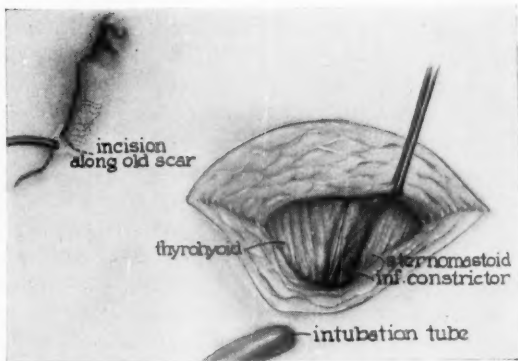


Fig. 3.—Drawing showing incision and approach to the arytenoid cartilage.

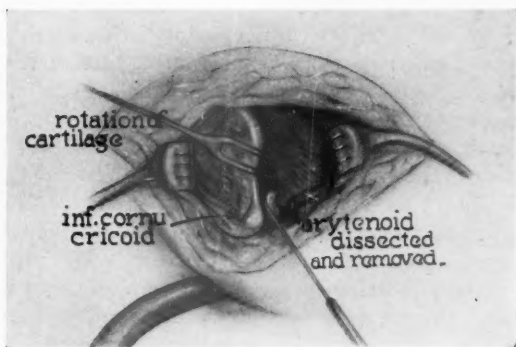


Fig. 4.—Drawing showing rotation of the larynx and exposure of the arytenoid cartilage.

moved. The result in both of these cases was satisfactory and the particle of cartilage did not react as a foreign body.

*Modification of Technique.* The operation is performed under intravenous sodium pentothal anesthesia. After preparation of the skin of the neck a sterile Waters-Guedel endotracheal catheter with a distendable cuff is passed through the tracheotomy opening. After inflation of the cuff there is no chance of blood leaking into the trachea. An incision is made through the old thyroidectomy scar, curving around the tracheotomy opening, and the skin flap is elevated until the residual pretracheal muscles and the larynx are identified (Fig. 3). An incision is made between the thyrohyoid and inferior constrictor muscles of the pharynx, along the posterior border of the lamina of the thyroid and extending down until the inferior cornu is disarticulated from the cricoid cartilage. The larynx can then be rotated upward and medially until the crico-arytenoid joint is identified by palpation. The arytenoid cartilage can then be retracted by small hooks and dissected out by subperichondral elevation of the muscle attachments (Fig. 4). After removal of the cartilage a No. 0 linen suture on a small, curved, round needle is passed through the fibers of the thyro-arytenoid muscle, the round needle is removed and a No. 3 straight Lane cleft palate needle is threaded on each end. The Lane cleft palate needles are brought through the posterior inferior third of the wing of the thyroid cartilage about 4 mm apart (Fig. 5a). Tension is exerted on the suture and the degree of abduction is checked by examining with an anterior commissure scope. When the cord is fixed in abduction the suture is tied over the thyrohyoid muscle (Fig. 5b). The incision along the posterior border of the thyroid cartilage is closed with interrupted catgut, a small cigarette drain is inserted and the skin incision is closed with skin clips or interrupted dermal sutures. The drain is removed on the third day and the skin sutures on the fifth. All patients are kept on penicillin for at least three days postoperatively. When the tracheotomy is closed elliptical incisions are made above and below the thyroidectomy scar and the opening into the trachea (Fig. 6a) and the previous scar is dissected out. The skin edges are undermined and the incision closed with interrupted dermal or skin clips (Fig. 6b).

#### ILLUSTRATIVE CASES

CASE 1.—Mrs. J. L., aged 44, was admitted to Hermann Hospital on April 12, 1946, because of shortness of breath. She had had a thyroidectomy in 1941, which was followed by a temporary loss of voice and the gradual onset of dyspnea. Since the operation she had been too ill to do her housework and had lost about 40 lb.

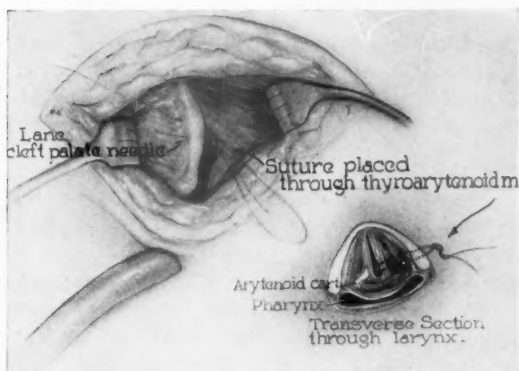


Fig. 5.—*a*. Drawing showing suture in thyro-arytenoid muscle being fixed through the ala of the thyroid cartilage. *b*. Diagrammatic drawing showing horizontal of section of suture.

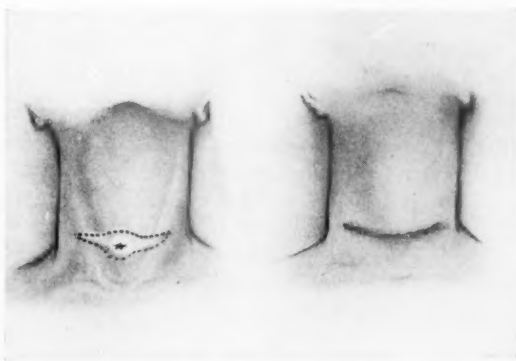


Fig. 6.—*a*. Drawing showing incision around tracheotomy opening with removal of scar. *b*. Drawing showing closure of incision.

in weight. She developed a cold two days before admission which made the dyspnea much worse. Examination of the larynx revealed both cords to be fixed in the midline. The patient was anemic and undernourished but presented no other abnormal findings except an acute upper respiratory infection. A low tracheotomy was performed on April 19, 1946. She was discharged on May 2, 1946, after the tracheotomy healed and she regained her strength and weight.

She was not seen again until January 21, 1947, when she was re-admitted because of infected granulations about the tracheotomy opening and cellulitis of the adjacent tissue of the neck. After débridement of the infected granulations and administration of penicillin, the infection subsided. On February 18, 1947, an arytenoidectomy was performed on the right side under intravenous sodium pentothal anesthesia. Woodman's modification of the Kelly operation was employed, the arytenoid was dissected out subperichondrally and a silk suture through the thyro-arytenoid muscle was anchored to the inferior cornu and the sternomastoid muscle. The incision was drained and closed with interrupted silk sutures. The drain was removed on the second day and the skin sutures on the fifth day. There was considerable postoperative swelling in the arytenoid area but the patient was able to swallow liquids the day after operation. She was discharged on March 5, 1947, and at this time there was an adequate breathing space, and she kept the tracheotomy tube plugged. Her voice was rather hoarse but was serviceable. On April 18, 1947, under local anesthesia, the tracheotomy was closed.

This patient has been able to resume her duties of a housewife and does not have any dyspnea after moderate exercise. Her voice has improved but is still low and husky. She has a basal metabolic rate of -26 and is very negligent in keeping her hypothyroidism stabilized. She has three scars on her neck (Fig. 1) but is so pleased to be able to discard her tracheotomy tube that these scars or the husky voice do not cause her any concern.

CASE 2.—Miss H. H., aged 27, developed hoarseness and later shortness of breath following a thyroidectomy in 1944. Her condition became progressively worse: she would have choking spells and spasms of coughing. She was advised that she had a bilateral abductor paralysis of the vocal cords but she refused tracheotomy. On June 11, 1945, an emergency tracheotomy was performed after she received a contusion of the neck in an automobile accident. The tracheotomy healed and she was fitted with a Tucker valvular tracheotomy tube which gave her a serviceable voice. At the time the



Fig. 7, Case 2.—Photograph (Miss H. H.) showing one scar after thyroidectomy, tracheotomy through old thyroid scar and arytenoidectomy.

thyroidectomy and tracheotomy were performed she was married but her husband was serving overseas. Soon after his return their marriage ended in a divorce which she attributed to the tracheotomy. She had a hypothyroidism which she kept stabilized by taking thyroid extract. The presence of the tracheotomy caused her considerable mental anguish but she was apprehensive about additional surgery.

On May 10, 1948, an arytenoidectomy was performed on the left side under intravenous sodium pentothal anesthesia. The tracheotomy had been placed just below the old thyroidectomy scar. A sterile endotracheal catheter with a distendable cuff was inserted into the tracheotomy and inflated to prevent leakage into the trachea. An incision was made through the old scar curving over the opening in the trachea. The skin, superficial fascia and platysma were elevated until the remnants of the pretracheal muscles and the larynx were identified. The crico-arytenoid joint was exposed by incising along the posterior border of the wing of the thyroid cartilage, according to the Woodman technique. The arytenoid was removed by subperichondral dissection. A No. 0 linen suture was passed into the substance of the thyro-arytenoid muscle and transfixed through the lamina of the thyroid cartilage. Checking by laryngo-

scopic examination showed the cord abducted and ample glottic space present. The muscles were approximated with interrupted No. 0 plain catgut sutures and the skin incision closed with clips. The patient was discharged from the hospital at the end of a week. After two weeks the patient wore the tracheotomy plugged day and night. On June 25, 1948, the tracheotomy tube was removed and elliptical incisions were made above and below the opening. The scars of both the thyroidectomy and tracheotomy were dissected out. The opening into the trachea was closed, the skin edges were undermined and the incision closed with clips.

This patient has adequate breathing space for any type of activity and her voice is only slightly lower than before the operation. There is only one scar on her neck (Fig. 7) and it is no more noticeable than any thyroidectomy scar. She has regained her self-confidence and has returned to work as a waitress, which she was before her thyroid operation.

#### COMMENT

This modification of technique employs both the principles of Kelly's operation and Woodman's approach. The horizontal incision through the previous thyroidectomy scar gives adequate exposure to work on either side of the larynx. It offers as much access as an incision along the anterior border of the sternomastoid muscle and the cosmetic results are much better. These patients have usually had several operative procedures on their necks and desire as little postoperative disfigurement as possible. This approach eliminates subsequent plastic surgery to remove unsightly scars.

The larynx can be mobilized as thoroughly as by any other procedure. Retraction and rotation of the larynx give good exposure of the crico-arytenoid joint. Transfixing the suture in the thyro-arytenoid muscle through the lamina of the thyroid cartilage draws the cord into the same position of abduction as Kelly obtains with his window technique. It also eliminates the difficulty of working through a small opening and preserves the wing of the thyroid cartilage to serve as an anchoring base for the vocal cord.

1304 WALKER AVENUE.

#### REFERENCES

1. Ballance, C.: Results Obtained in Some Experiments in Which the Facial and Recurrent Laryngeal Nerves Were Anastomosed with Other Nerves, *Brit. M. J.* 2:349-354, 1924.
2. Ballance, C., and Barnes, E. B.: Anastomosis of Recurrent Laryngeal Nerves to Phrenic Nerves. Some Recovery of Function, *Brit. M. J.* 2:158-159, 1927.
3. Clerf, L. H.: Bilateral Paralysis of the Larynx. Surgical Treatment. Read before the meeting of Am. Acad. Ophth. and Otolaryng., Chicago, Ill., October 14, 1948.

4. Frazier, C. H.: Treatment of Paralysis of Recurrent Laryngeal Nerve by Nerve Anastomosis, *Ann. Surg.* 79:161-171 (Feb.) 1924.
5. Frazier, C. H.: Anastomosis of the Recurrent Laryngeal Nerve with the Descendens Noni in Cases of Recurrent Laryngeal Paralysis, *J. A. M. A.* 83: 1637-1641, 1924.
6. Frazier, C. H., and Mosser, W. B.: Treatment of Recurrent Laryngeal Nerve Paralysis by Nerve Anastomosis, *Surg., Gyn. and Obst.* 43:134-139 (Aug.) 1926.
7. Galloway, T. C.: Modified Kelly Operation for Abductor Paralysis, *Arch. Otolaryng.* 34:1197 (Dec.) 1941.
8. Hoover, W. B.: Bilateral Abductor Paralysis: Operative Treatment by Submucous Resection of the Vocal Cord, *Arch. Otolaryng.* 15:337-355 (Mar.) 1932.
9. Jackson, C.: Ventriculocordectomy. A New Operation for the Cure of Goitrous Paralytic Stenosis, *Arch. Surg.* 4:257-274 (Mar.) 1922.
10. Kelly, J. D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles, *Arch. Otolaryng.* 33:293-304, 1941.
11. Kelly, J. D.: Some Problems in the Surgical Treatment of Bilateral Abductor Paralysis of the Larynx, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 53:461-468 (Sept.) 1944.
12. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis. Preliminary Report, *J. A. M. A.* 112:814-823 (Mar.) 1939.
13. Lahey, F. H.: Suture of the Recurrent Laryngeal Nerves for Bilateral Abductor Paralysis, *Ann. Surg.* 87:481-484, 1928.
14. Lahey, F. H., and Hoover, W. B.: Injuries to the Recurrent Laryngeal Nerve in Thyroid Operations, *Ann. Surg.* 108:545, 1938.
15. Lederer, F. L., and Howard, J. C.: Wartime Laryngeal Injuries, *Arch. Otolaryng.* 43:331-343 (Apr.) 1946.
16. Lore, J. M.: A Suggested Operative Procedure for Relief of Stenosis in Double Abductor Paralysis: An Anatomic Study, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 45:679-686 (Sept.) 1936.
17. McCall, J. W., and Gardiner, F. S.: A Simplified Operation for Bilateral Abductor Paralysis, *Laryngoscope* 53:307-311 (May) 1943.
18. Morrison, L. F.: Bilateral Paralysis of Abductor Muscles of the Larynx. Report of Seven Patients Treated by the Method Outlined by Dr. Brien T. King, *Arch. Otolaryng.* 37:54-61, 1943.
19. Morrison, L. F.: Further Observation on the King Operation for Bilateral Abductor Paralysis, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 54: 390-408, 1945.
20. Orton, H. B.: Surgical Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Larynx, *Laryngoscope* 53:709-716 (Nov.) 1943.
21. Seed, L.: The King Operation for Bilateral Abductor Paralysis of the Vocal Cords, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 51:66-78 (Mar.) 1942.
22. Shirer, J. W.: Modification of the King Operation for Bilateral Vocal Cord Paralysis, *Ann. Surg.* 120:617-622, 1944.
23. Thornell, W. C.: Intralaryngeal Approach for Arytenoidectomy in Bilateral Abductor Vocal Cord Paralysis. Read before the meeting of Am. Acad. Ophth. and Otolaryng., Chicago, Ill., Oct. 13, 1948.
24. Tucker, G.: Valvular Tracheotomy Tube, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 43:1128-1129 (Dec.) 1934.
25. Woodman, D. G.: A Modification of the Extralaryngeal Approach to Arytenoidectomy for Bilateral Abductor Paralysis, *Arch. Otolaryng.* 43:63-65 (Jan.) 1946.
26. Wright, E. S.: The Kelly Operation for Restoration of Laryngeal Function Following Bilateral Paralysis of the Vocal Cords, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 52:346-337 (June) 1943.

## IX

### MALIGNANT MELANOMA OF THE NOSE AND SINUSES

JOSEPH G. SCHOOLMAN, M.D.

AND

HAROLD W. ANDERSON, M.D.

CHICAGO, ILL.

One of the most enigmatic, in the action and reaction of malignant neoplasms, is the group classified as melanoma. The greatest portion of our present day knowledge concerning the histogenesis, clinical behavior and therapeutic measures employed in combating this lethal tumor has been, in the main, accumulated during the latter part of the nineteenth and first third of the twentieth centuries. Contributions by investigators during the past 15 years have added little to the original data contributed by the investigators of the previously mentioned period.

From the long existent literature, certain generalizations have been noted. These, with the passage of time, have been accepted without the original qualifications. From corollary they have become law. The objective of the authors is to credit what is already known about melanomas, to distinguish theorem from deduction and to emphasize the fact that no two cases have exactly the same histogenesis, clinical course or therapeutic response. Several surmises and dogmatisms concerning the behavior of melanoma in regard to its incidence, relation to race, age, sex and site of origin are reviewed.

*Definition.* Melanomas are tumors originating from melanoblasts. These are cells which actually produce melanotic pigment or are capable of doing so. They are not to be confused with the melanophores which are histocytes and do not in themselves produce pigment but may contain pigmented material by phagocytosis.

Melanin is a characteristic feature of melanomas. The amount of pigment present varies considerably both in the same tumor and between the primary and the metastatic tumors. Certain parts of the tumor may contain more pigment than others, and in some forms of melanoma, pigment may be absent. The latter are known as amelanotic melanomas.

---

From the Departments of Otolaryngology, University of Illinois College of Medicine, Mount Sinai Hospital, and Provident Hospital, Chicago, Illinois.

*Histogenesis.* The theories applied to the origin of melanomas are as follows:

1. Exclusively from mesodermal chromatoblasts.
2. Exclusively from epithelial cells and epithelial chromatoblasts.
3. From nevus cells in epithelium and mesodermal chromatoblasts in choroid and meninges.
4. From endothelial cells of blood and lymph vessels or from nerve trunks.
5. From chromatoblasts in relation to tactile corpuscles and nerve cells forming the end apparatus of cutaneous sensory nerves.

These theories concern the origin of the melanoblast. Is it a separate cell entity? Is it an epithelial or a mesenchymal element, or is it common to both categories? Masson in 1926 offered the theory that skin nevi develop from neurogenous cells which are differentiated into sensory cells. Ringertz believes that Masson's theory of the neurogenous nature of nevus formations may enable us to consider melanoma as a genetic unit. Some investigators have felt that the Langerhans cells which lie in the deeper parts of the epithelial layer are melanoblasts and their invasion of the corium gives rise to nevus cells. They feel that these cells are of epithelial origin and therefore the tumors which they produce are epithelial tumors—melanocarcinomas or nevocarcinomas. It is more commonly accepted that the melanoblast is a mesenchymal element. It is thought to occur in the corium, in the choroid of the eye, in the leptomeninges and in the submucosa of the respiratory and digestive tracts. Tumors taking origin from this cell would constitute mesenchymal melanoblastomas or melanosarcomas. It is understandable that this tumor, which may represent a group rather than a single entity, should show variability in its clinical picture and its response to therapy.

The most recent and most acceptable theory is that the chromatoblasts arise from cells which have migrated from the neural crest in early embryonal development.

*Histology.* The histologic picture varies, depending upon whether or not the growth arose from a previously existing nevus or from an otherwise "normal" tissue. The histologic details which characterize malignant transformation of a nevus are the disintegration of the architectural arrangement of the cellular nests seen in nevi; an increase in pigmentation, more particularly in the periphery, and an inflammatory reaction in the stroma. In those tumors arising in previously "normal" tissue, mitosis sometimes occurs. In

the early stages one finds a proliferation of the cells in the deeper malpighian layers. The germinative layer is disintegrated, and large globular or irregular cells with abundant protoplasm are present. They may form nests surrounded by the malpighian cells. There is an associated inflammatory reaction of the connective tissue elements. In the latter, however, the pigment has been included by phagocytosis.

The distribution of the pigment is not always visible but can at times be demonstrated by the impregnation of silver, following the Bloch's dopa reaction. From the diagnostic point of view, this test has only limited value in that not all pigmented regions give a positive reaction and some investigators feel that the reaction is not specific for melanogenous substances. Ringertz, in analyzing the pigment in his group of cases, stained for iron and also used Masson's melanin stain. He found that a small part of the tumor's pigment was due to hemosiderin which he explained as due to the vascularity of the tumor and the multiple hemorrhages so often associated with melanomas. Masson's silver reducing method of staining reveals melanin as black while the hemosiderin retains a brownish color.

The histologic appearance of a metastatic tumor does not always coincide with that of the primary lesion, especially as regards pigmentation, thus an amelanotic primary growth may produce a highly pigmented metastasis. If one should examine the cutaneous surface of an individual, it can be demonstrated that 20 or more pigmented cutaneous lesions (nevi) are present. It may be more difficult to show this on a dark skinned individual, particularly the negro. These benign lesions offer no problem unless they are considered precancerous or are undergoing malignant transformation.

*Race Incidence.* The question of incidence of melanoma in different races has been considered. Thorough investigation of literature describing geographic pathology and racial differences in pathology fails to give us any enlightening information on the melanoma problem. The following generalizations are made: 1.) Malignant melanomas are less common in negroes than in whites. 2.) Regional metastases are unusual in negroes and simple local excision as a rule is sufficient to effect a cure. 3.) Lymph node metastasis is of extremely common occurrence in malignant melanoma of the skin and no group (save possibly children and negroes) enjoys even a relative immunity in this regard. The cases observed in our clinic show that half occurred in caucasians and half in negroes. This observation suggests that the conclusion that negroes are less susceptible is not valid.

Two hundred and ten normal individuals were examined by the authors to determine whether there was any relationship between the normal pigmented areas of the upper respiratory-digestive tracts to the site of origin of melanomas observed in our cases.

Conclusions drawn from the above observations speak against the possibility that malignant melanoma may arise from what is termed precancerous melanosis, a condition characterized by melanotic spots which occur in individuals and remain stationary or dormant for years and then supposedly turn malignant. One must also bear in mind where benign and malignant forms occur on the

## RACE INCIDENCE

		CAUCASIAN (50)		NEGRO (160)		
			50	50	50	10
			DARK SKIN	BROWN SKIN	LIGHT SKIN	ALBINO
Oral Cavity						
Gingivae	O	Ma	Mo	Mo	Mo	N
Palate	O	Mo	O	O	O	O
Nose						
Vestibule	O	Mo	O	O	O	N
Internal	N	N	N	N	N	N
Oropharynx	N	O	O	N	N	N
Nasopharynx	N	N	N	N	N	N
Mesopharynx	N	N	N	N	N	N

O: Occasional melanotic area; Mo: Moderate melanotic area;  
Ma: Marked melanotic area; N: no melanotic area.

external surfaces of the body. Malignant melanoma occurs frequently on the feet and genitalia where nevi are rare. This fact also enforces our suspicion that melanotic areas are not necessarily the sites of origin of malignant melanomas.

*Clinical Classification of Melanomas.* 1. Benign. Pigmented nevi—cutaneous origin.

2. Malignant. (a) Malignant transformation from pigmented cutaneous nevi; (b) choroid; (c) meninges; (d) melanomas, origin still undetermined.

It is the 2d group with which this paper is most concerned and from which all our cases are categorized. Investigation of the literature reveals that most data discuss melanomas arising from cutaneous origin. The clinical importance of nevi lies only in the possibility of their transformation into malignant melanomas.

Ringertz added six cases of primary malignant melanoma in the nose and sinuses to 35 others reported in the literature. During the past three years the incidence of melanoma of the upper respiratory tract has come to the attention of our Department of Otolaryngology. Review of the active and inactive files of the Tumor Clinic revealed the following cases of malignant melanoma:

Paranasal sinuses, 2

Nasal septum, 2

Nasal vestibule, 2

#### AGE INCIDENCE

YEARS	30-39	40-49	50-59	60-69	70-79	80-89
32 cases from the literature	3	4	14	5	4	2
Ringertz' cases			1	2	3	0
Our cases		2	2	2		
Total	3	6	17	9	7	2

The lesions arising in the nasal vestibule are, to the best of our knowledge, the first to be reported in the literature. There were no cases on record of lesions involving the upper digestive tract. It is not known whether the respiratory epithelium (specialized mucous membrane) has some innate property, anatomical or chemical, which predisposes to malignant melanoma.

The age incidence of our cases is substantially in agreement with that indicated in earlier reports. The sex incidence, while not in agreement with the finding of Ringertz, is similar to the findings for the total cases reported.

*The Therapeutic Approach.* Before considering technique in management of malignant melanoma or indeed of any of the cancerous group of diseases, we must emphasize diagnosis. To diagnose the disease we must have a conscious awareness of its existence and of the probability of its presence. Painstaking examination must

be routine regardless of the specific complaint of the patient. The chance of cure is greater if the lesion can be eliminated before it overtly manifests itself. The elimination of the disease must be total. In cancer therapeutics the primitive doctrine of kill or be killed is especially true. Action must be swift, decisive, and heroic. To temporize is to surrender without battle. To economize in the scope of our surgical or radiological attack is to fall short of success. To consider post-therapeutic deformity is to lose the battle. Here most certainly the more one saves the part, the more surely one has lost the whole.

Our record is poor. Our weakness has been conservatism. The facts, however, may be of value to ourselves and to our colleagues. The authors therefore propose to offer in this paper the methods employed by them and their associates in dealing with malignant melanoma of the nose and sinuses.

## SEX INCIDENCE

	MALE	FEMALE
53 cases from the literature	16	19
Ringertz' cases	1	5
Our cases	3	3
Total	20	27

*Variants in the Therapeutic Measures Used in Malignant Melanoma.* Many clinicians believe that pigmented tumors should not be biopsied, on the grounds that such trauma will cause immediate dissemination with widespread or generalized metastases. Ewing states that simple traumas may excite quiescent nevus cells to overgrowth and renewed pigment production, developing a malignant localized tumor. The overlying epidermis soon participates in this tumor, developing pigment, the presence of which still further stimulates growth and often the extension of the tumor process along the basal cells of previously normal rete pegs. From both of these sources, actively proliferating epithelial cells bearing pigment and devoid of fibrils become disseminated by lymph and blood vessels.

Evans and Lencitta conclude that certain destructive therapeutic procedures now employed act in a similar manner as traumas, pro-

ducing degeneration of pigmented nevi into malignant processes or dissemination of already malignant melanomas to distant parts of the body. Two such methods employed are electrogalvanization and the sparking by high frequency current, both of which, regardless of how carefully applied, act in the form of minute traumatic impulses to the tissues exposed. The same criticism applies to the use of electrocautery and electrocoagulation, if unskillfully used on pigmented moles or on tumor formations. It seems that trauma disturbs the equilibrium which exists between the potentially malignant pigmented cells and the defense mechanism of the surrounding tissues, a phenomenon commonly known as the quiescent state, bringing about a sudden release of malignant tendencies, either to local growth or more commonly to extensive spread with rapid lethal outcome. The question arises, is surgical excision by knife a better or safer procedure? It is conceded that the majority of investigators favor excision of the pigmented moles and melanomas with a wide margin and without injury to the pigmented area.

F. Christopher states that excision is probably the safest method of removing moles and that at least one-eighth inch of normal tissue surrounding the melanoma should be removed in one block without traumatizing the lesion in any way. If local anesthesia is used, the injection should be well away from the lesion. Francis G. Woods advocates excision, early, wide and including regional nodes. Bloodgood, not so sure of the use of surgical removal, advises knife in benign lesions, cautery for malignant.

Taylor and Nathanson state that the likelihood of metastases is increased by unsuccessful attempts at local cure. They feel that local recurrence, regardless of the original treatment employed, increases the incidence of node metastasis. They conclude that the use of radium, cautery, electrocoagulation, or desiccation are particularly likely to result in local recurrences; and that the most effective local therapy is wide surgical excision.

Handley recommends not only removal of nodes, but also the intervening lymphatics, which are also implicated. Of 200 malignant mole operations performed by Bloodgood in a period of 30 years, only one was a five-year cure, and according to McEven, this patient had some postoperative roentgen therapy which, in his opinion, produced this one cure.

Wigby and Metz reported that one patient with widespread metastasis to skin and lung was given irradiation to the pituitary gland. The result was the elimination of all lesions microscopically

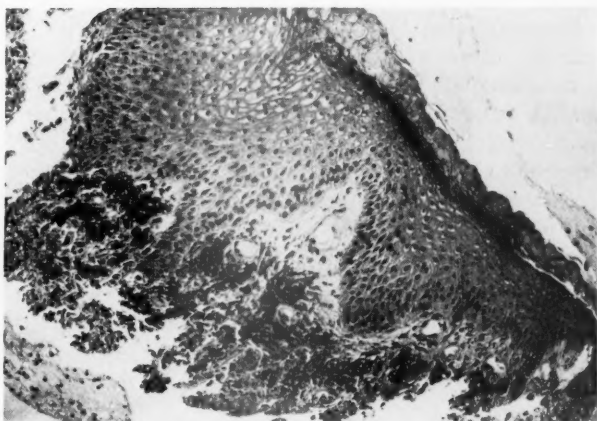


Fig. 1, Case 2.—Photomicrograph showing keratotic squamous epithelium overlying connective tissue, containing many brown pigment filled cells and fusiform cells with pink cytoplasm and dense blue fusiform nuclei.

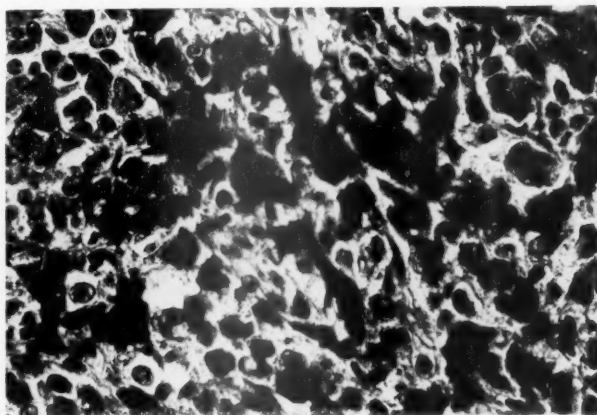


Fig. 2, Case 2.—Photomicrograph showing actively proliferating tumor cells, frequently spindle shaped and often polyhedral. Cytoplasm is often abundant and contains large quantities of granular brown pigment. Some cells lack the pigment entirely. The nuclei are large, show anisocytosis and great variation in staining reaction. Mitotic figures are fairly plentiful.

and roentgenographically. They concluded that the intermediate lobe of the pituitary produces a hormone (secretion) that is apparently necessary to growth and reproduction of the melanoblast and also for abnormal cells to remain viable. They also state that the spleen seldom receives malignant metastasis when all other organs are involved, concluding the spleen contains some physiologic chemical antagonist to the growth of melanotic tissue. They also treated a series of cases of clinically malignant melanomas with hypermassive doses of irradiation and claim excellent results. The hypermassive doses represent an amount of roentgen rays which produces a cytocaustic action of the tissue exposed, and is equal to several erythema doses.

Others believe melanomas are 100% radioresistant and only suggest irradiation as a psychopalliative measure.

#### REPORTS OF CASES

CASE 1.—T. G., a 68-year-old white female, weight 127 lb., was first seen in the Department of Otolaryngology on October 12, 1945, with a history of pain and swelling involving the right side of the nose for two and one-half months. Examination disclosed moderate swelling of the right ala and obstruction of the right nasal cavity by a mass. Grossly the lesion appeared pigmented and a clinical diagnosis of melanoma of the vestibule was made. Biopsy performed October 12, 1945, was reported by the Pathology Department as malignant tumor, unclassified. A repeat biopsy was requested and was performed October 26, 1945. It was reported as malignant melanoma. Roentgenograms on October 20, 1945, revealed no definite evidence of neoplasm, or disease in the paranasal sinuses. On October 26, 1945, an additional mass was noted which grossly appeared to be connected to the right maxilla sublabially near the nasal spine. Because of its hardness aspiration biopsy was impossible as the needle could not be pushed through the cortex. This mass was not visualized on x-ray examination. Surgery was considered, but because of the patient's age and general condition including a known parkinsonism, it was not advisable to operate. Despite the accepted dictum that melanomas are radioresistant it was decided to try irradiation as a palliative form of therapy. A Picker-Villard machine was used with the following radiation factors: 200 kv, 0.81-mm copper, half value layer 20 ma, 50-cm focal skin distance, 50 r per minute, 4.5-cm field to right nostril and canine fossa. A total of 4500 r, in 22 divided doses, was administered between November 23, 1945, and January 25, 1946. Supplementary interstitial irradiation was deemed advisable and on March 22, 1946, 15 gold radon seeds of 1.5-millicurie strength each



Fig. 3, Case 2.—A 45-year-old negress with malignant melanoma of the nose as seen prior to therapy.



Fig. 4, Case 2.—Malignant melanoma of the nose following irradiation and nitrogen mustard therapy.

were implanted into the tumor mass through the skin. The total interstitial dose was 3000 millicurie-hours.

Despite irradiation therapy the lesion increased in size. On April 5, 1946, the patient complained of tenderness in the area treated and of a productive cough. There was a marked postradiation atrophy of the entire upper lip. Roentgenogram of the chest disclosed numerous irregular infiltrating densities throughout both lung fields which were considered metastatic lesions. The patient was last seen in our clinic April 19, 1946, and she expired at home May 21, 1946. No autopsy was performed. The patient had only a 6-lb. weight loss during her period of observation in our clinic.

CASE 2.—L. C., a 45-year-old negress entered our clinic September 20, 1946, with a huge tumor originating in the nose which had been present and growing for one year. The lesion had destroyed the normal landmarks of the nose and protruded into the mouth through the palate; it filled the nasal cavity and obstructed the nasopharynx. X-ray examination of the paranasal sinuses did not reveal ethmoid involvement. Biopsy revealed malignant melanoma. The lesion was classified as inoperable and irradiation therapy was recommended. On December 4, 1946, internasal sloughing of the tumor mass was noted during the first course of irradiation. The factors during the first course were as follows: General Electric Maximar 400 kv, 2.75-mg copper half value layer, 200 r per treatment, 3000 r each of four 4 x 5-cm portals, superior nasal left anterior oblique, right anterior oblique and inferior nasal fields. Treatment extended from October 18, 1946, to January 17, 1947. On January 8, 1947, it was noted that the mass had regressed approximately 50%. At the end of her first course of irradiation residual tumor was noticed in the floor of the nose and a course of nitrogen mustard was recommended. This course extended from April 5, 1948, to May 4, 1948, and a total of 265 mg was administered in six divided intravenous doses. Involvement of the anterior portion of the nasal vestibule was noted on March 19, 1947, and an additional course of irradiation was recommended. The factors for this course were as follows: Picker-Villard, 200 kv, 0.75-mm copper, 2-mm aluminum, 0.95-mm copper half value layer, 25 ma, 50 cm focal skin distance, 1800 r each of two portals, right and left direct nasal fields, from April 5, 1948 to May 4, 1948.

A second course of nitrogen mustard was given from May 28, 1947, to June 9, 1947. A total of 28 mg was administered in four divided doses. Gastro-intestinal upset was noted throughout the course. A third course of nitrogen mustard extended from September 22, 1947, to October 6, 1947. A total of 28 mg was ad-

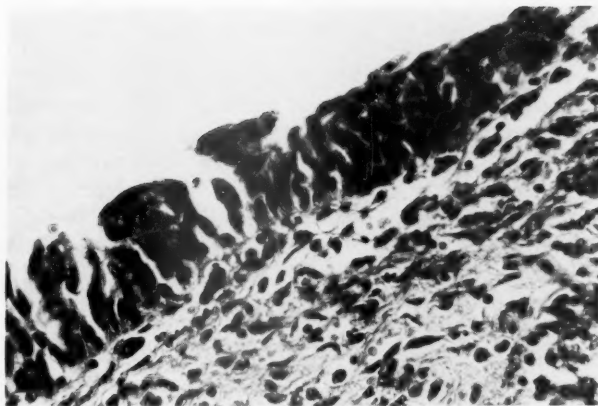


Fig. 5, Case 3.—Photomicrograph showing respiratory type of mucous membrane with cells containing golden brown pigment just deep to the basement membrane. The deeper tissues are completely replaced by masses of anaplastic tumor cells which are growing almost solidly. These cells are polygonal in shape for the most part with abundant cytoplasm and large vesicular nuclei. Anisocytosis, hyperchromatism, and atypical mitosis are present. In areas these tumor cells are spindle shaped. Rarely, brown pigment is seen in the cytoplasm of these cells.

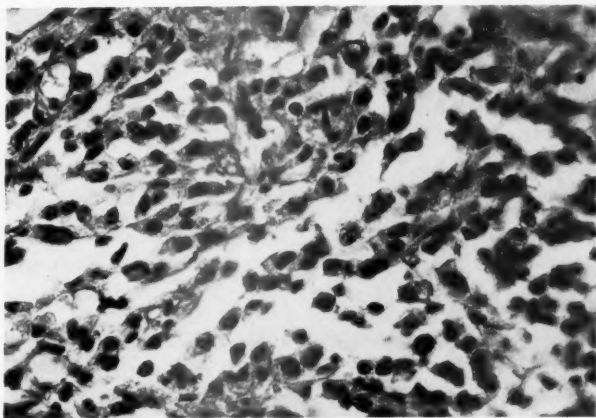


Fig. 6, Case 3.—The tumor consists of dense sheets of pleomorphic cells with eosinophilic cytoplasm and darkly staining rounded nuclei. Some of them contain yellow-brown pigment. Occasional mitotic figures are seen.

ministered in four divided doses. Again gastro-intestinal symptoms were experienced by the patient.

At present the patient's general condition is relatively good despite residual tumor in the nasal cavity. Local exenteration was considered, but the development of a solitary pulmonary metastasis prevented this and the patient was transferred to our chemotherapy section for treatment with one of the experimental drugs under investigation. The patient is maintaining her original weight. The blood picture remains within normal limits. There is, however, a glycosuria which is being controlled by the metabolic service. Except for an offending odor from the nasal cavity, the patient is apparently comfortable and without complaint.

CASE 3.—C. M., a 51-year-old white male entered our clinic October 15, 1946, presenting a deep red mass occluding the left nostril which could also be seen by posterior rhinoscopy but did not extend into the epipharynx. A metastatic node was present in the right submaxillary area. Biopsy of the nasal mass was reported as malignant melanoma. The diagnosis from a previous biopsy by a private physician, February 16, 1945, could not be obtained. At another Tumor Clinic a biopsy was performed in February, 1945 and reported as squamous cell carcinoma. The lesion was treated there as follows: X-ray therapy was given from February 28, 1945, to March 22, 1945. A total dose of 4,000 r units was given on the left side and 1500 r units on the right side, directed toward the nose. The factors were 400 kv, 5 ma, 5-mm copper plus 1-mm aluminum filtration, 75-cm focal skin distance and 4.5 x 3.5-cm portal on the left and 4.5 x 3-cm portal on the right; 20 doses to left, 6 doses to right.

On June 21, 1945, under local anesthesia, one 3.1-mg, two 2.2-mg and one 1-mg removable platinum radium needles were inserted into the residual lesion of the nose. These were allowed to remain in for 101½ hours. A total dose of 862.75 mg-hr. was given.

At the time of his admission to our Clinic, his weight was 147 lb. On November 7, 1946, the patient had a radical resection of the tumor mass of the nose and accessory sinuses, through a modified Moure incision and an enlarged Denker operation. Residual was left in the region of the cribiform plate which had failed to show on roentgenography, October 17, 1946. On January 15, 1947, the submaxillary node on the right side was removed. Sections revealed metastatic involvement.



Fig. 7, Case 4.—Recurrent malignant melanoma in nose two years after subtotal resection of maxilla.

The patient was sent to the X-ray Department for irradiation therapy to the residual tumor tissue, from February 7, 1947, to May 2, 1947. Picker-Villard machine was used, 200 kv, 0.81-mm copper half value layer, 20 ma, 50-cm focal skin distance, 46 r per minute, 6 x 8-cm field, a total of 3900 r, 300 r per treatment, to the right submaxillary node area. A total of 3200 r, 5 x 6-cm field, 300 r per treatment, to the left posterior submandibular node. General Electric Maximer 400 kv, 2.75-mm copper half value layer, 5 ma, 50-cm focal skin distance, 45 r per minute, 4.5-cm portal to anterior ethmoid plate. A total of 3100 r, 200 r per treatment.

The patient developed a secondary anemia due to hemorrhage from the nose and received transfusions and supportive treatment.

A course of nitrogen mustard was advised. A total of 24.4 mg was given from June 13, 1947, to June 20, 1947, in four divided doses. Gastro-intestinal symptoms were experienced by the patient.

The patient became weaker, the anemia persisted, and locally the submaxillary node along with a pre-auricular node and nasal lesion appeared to be increasing in size. Despite the downhill picture, another course of nitrogen mustard was given from July 23, 1947, to August 1, 1947. A total of 21.1 mg in four divided doses was administered. The patient complained of cough, and x-ray examination of the chest was advised to see if there was any metastatic lesion in the chest. He refused to be subjected to the exam-

ination and was advised to return at a later date. We were notified that the patient expired at home on August 23, 1947. No autopsy was performed.

CASE 4.—M. R., a 65-year-old negress, weight 122 lb., entered our clinic February 25, 1947, with history of nasal congestion and bleeding from the right nostril of one year's duration. Anterior rhinoscopy revealed a pigmented mass just posterior to the nasal vestibule on the right side. X-ray films of the paranasal sinuses revealed involvement of the right maxillary sinus. The ethmoid area appeared roentgenographically negative for extension. Biopsy revealed malignant melanoma.

A radical resection of the right maxilla and ligation of the external carotid was advised. On March 11, 1947, the above procedure was performed, at which time, grossly, the entire mass was removed. After healing of the operative defect, it was noted that residual or recurrence was present along the soft palate. Cauterization of this area was performed on April 12, 1947.

Frequent inspection from April, 1947, to April 1948, failed to show any local recurrence. On April 2, 1948, there was noted a submandibular node on the right side which was resected and reported as metastatic malignant melanoma. There was also noted in the operative defect, melanotic areas involving the ramus of the nasal septum and the remains of the right lateral nasal wall. These areas were desiccated with diathermy. Early in 1949 local recurrence was noted. Investigation revealed widespread extension. The patient was transferred to the chemotherapy section for further treatment.

CASE 5.—L. D., a 45-year-old negro, weight 247 lb., appeared at our clinic May 7, 1946, with a history of nasal congestion of two years' duration. He had consulted a private physician in October, 1944, and was referred to a local institution for removal of a mass which was attached to the nasal septum on the left side. The mass was removed and its base on the septum was cauterized by electrocautery. Examination revealed malignant melanoma. Frequent inspection to June 6, 1946, failed to reveal any recurrence. A septal perforation resulted from surgery. On June 6, 1946, there was noted a recurrence along the border of the septal perforation. This recurrence was removed under local anesthesia, with the electric diathermy cutting current. Several inspections, up to February 14, 1947, failed to show any local recurrence. On this date a swelling of the dorsum of the nose was noted, which had a bluish discoloration. Aspiration biopsy revealed malignant melanoma. The patient did not return to our clinic until May 9, 1947. He stated he

had been ill in another hospital, and had been treated for a chest condition. He had had a 20-lb. weight loss during the past six weeks. X-ray films of his chest revealed bilateral pulmonary metastasis, with right hydrothorax.

A course of nitrogen mustard was administered from May 19, 1947, to May 28, 1947. Forty-five milligrams in five divided doses was given. The patient experienced gastro-intestinal upset. The blood picture on July 14, 1947, was reported as within normal limits. A second course of nitrogen mustard was given from July 14, 1947, to July 21, 1947. A total 45 mg was administered in five divided doses. The usual gastro-intestinal symptoms were experienced.

The patient returned to our clinic September 8, 1947, stating that he had been hospitalized at another local hospital on July 31, 1947, where he received x-ray treatment to the chest because of his increasing dyspnea. Correspondence with the x-ray therapist at this institution gave the following information. Sixteen treatments were given between August 13, 1947, and September 2, 1947. Two anterior chest portals were treated alternately with two posterior chest portals, two fields, being treated daily. This amounted to 3000 r (skin per portal). Technical factors used were 200 kv, 50-cm target skin distance, 0.5-mm copper and 1-mm aluminum filter, 0.85-cm copper half value layer. X-ray films of the chest on September 8, 1947, revealed an increase in the size of the previously noted metastatic masses. We were notified by relatives on September 23, 1947, that the patient was too weak to return to our clinic for further care, and on October 6, 1947, that he had expired at home. No autopsy was performed.

CASE 6.—B. S., a 52-year-old negro, weight 178 pounds, appeared at our clinic July 25, 1947, with a history of having had a removal of the cartilaginous septum because of a mass which caused nasal symptoms and was diagnosed as malignant melanoma, grossly and microscopically. Examination revealed recurrence of melanoma along the border of the operative defect. A more radical procedure was advised and performed, September 16, 1947, by the Denker method. The bony nasal septum, the right lateral wall and inferior turbinate were removed.

On September 30, 1947, a polypoid mass was noted in the right antrum. This mass remained quiescent until December 23, 1947, when the patient complained of frequent attacks of epistaxis. It was recommended that another radical procedure be performed with removal of the hard palate. This was performed March 10, 1948.

At this time the patient had had a 21-lb. weight loss, but was without demonstrable metastases. On April 23, 1948, a 2 x 3 x 1-cm recurrence was noted in the lateral portion of the right antrum. This mass was cauterized by electrocautery, May 7, 1948. On May 21, 1948, residual in the antrum measured 5 x 3.5 cm and a metastatic node was present. Irradiation was recommended, and on June 4, 1948, breakdown of the epidermis overlying the cervical nodes was noted. We were informed by the X-ray Department that the amount of irradiation to date is negligible. Films of the chest do not reveal evidence of metastasis to the lung fields or bony thorax.

#### CONCLUSIONS

1. Six additional cases of malignant melanoma of the nose and sinuses are added to the 38 previously reported in the literature; two of these are, to the best of our knowledge, the first reported as originating in the nasal vestibule.

2. We have not found the tumor less frequently in the negro than in the white.

3. Malignant melanoma should not be considered radioresistant. Some tumors are affected by irradiation either primarily or secondarily due to interference with their blood supply.

4. Biopsy, cautery and diathermy seem to stimulate metastasis. Wide excision with the cold knife or irradiation appear to be the methods of choice.

5. The prognosis in malignant melanoma is poor.

1853 WEST POLK STREET.

X

SALIVARY GLAND TUMORS OF THE SUBMAXILLARY  
GLAND ASSOCIATED WITH CALCULI

EPHRAIM L. MANNING, M.D.

DAVENPORT, IOWA

AND

MAURICE A. MICHAEL, M.D.

LEBANON, PA.

On diagnosing a neck swelling as an affection of the submaxillary gland, we first think of an obstructing calculus or an infection, or both, and usually dismiss the diagnosis of tumor quickly, especially if a calculus can be demonstrated by palpation and x-ray examination. Two recent cases brought up the point that one must consider salivary gland tumor (so-called mixed tumor) of the submaxillary gland even if calculi can be demonstrated.

Very much has been written regarding the pathology and possible pathogenesis of salivary gland tumors and it would be advisable to briefly review this as well as to consider those matters regarding gland calculi.

Cheyne, Teicke and Horne<sup>1</sup> in their detailed review of salivary gland tumors state that the term "mixed tumor" applied to these neoplasms was originally proposed by Minssen in 1874, but that they are not teratomas since teratomas possess definite tissues forming atypical structures and rudimentary organs. These neoplasms are generally believed to be derived from epithelium and thus are not "mixed tumors."

As the above writers state, the main element of the salivary gland tumor is a polymorphous epithelial cell which forms into strands, alveoli and tubular structures or masses closely arranged or scattered in the surrounding tissue. The cell itself may be round, stellate, cuboidal, columnar or spindle shaped. Other than the cell the most characteristic structure is an excess of mucoid tissue which

---

Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

intermingles with the supporting stroma and is believed to be a product of the neoplastic cells.

The theories regarding histogenesis of the tumor, include:

1. *The embryonic theory*, which states that embryonic rudiments pinched off in the region of the salivary glands during development of the head, give rise to mixed tumors. Cohnheim in 1882 felt that they were derived from the cartilaginous parts of the bronchial arches. He, of course, thought of it as a mixed tumor with the epithelial remnants acquiring the features of glandular or squamous epithelium and the mesenchymal remnants acquiring those of simple connective tissue or any of its derivatives such as cartilage or bone. The chief refutation for this theory is, that known branchiogenic tumors are more teratomatous.

2. *The mesenchymal theory*, stating that the tumors arise from the connective tissue of the salivary glands with the epithelial-like structures as vestiges of the glands.

3. *The epithelial theory*, the most accepted at the present time, which states that they are derived from normal salivary gland tissue. The salivary glands develop from ectoderm of the primitive oral cavity and it is argued, both that the tumors may arise from embryonal tissue or from adult epithelium, which dedifferentiates.

4. *Ectomesodermal rests*, which is the idea of embryonal mesenchyme derived from ectoderm with misplacement of these rests. This includes Hellwig's theory<sup>2</sup> that they are derived from misplaced elements of the notochord.

5. *The endothelial theory*, stating that the tumors are derived from lymphatic endothelium.

Salivary gland tumors occur at all ages, with an average age of onset apparently of 35 to 40 years, and last on the average about six to nine years. They occur about equally in males and females and in the salivary glands occur chiefly in the parotid gland with, in order of frequency, next the submaxillary, and then sublingual gland. However, they also appear in the palate, cheek, lips, tongue, pharynx, and nares.

The usual history is of a previously quiescent nodule in or over the periphery of one of the larger salivary glands, growing slowly, taking about five years to reach the size of a walnut and about ten years to reach the size of a lemon. These tumors have a marked tendency to recur after operation because of the difficulty of removing all portions of the tumor, especially in the parotid gland. Malignant types of these tumors do occur and invade adjacent

structures, and there have been many cases of metastasizing mixed tumors reported although this occurs infrequently.

Salivary gland tumors are resistant to irradiation and surgical excision is the accepted treatment. It is interesting to note the relative frequency of occurrence of the tumors in the parotid, submaxillary and sublingual glands as found by various writers.

	PAROTID	SUBMAXILLARY	SUBLINGUAL
Ahlbom	286	39	3
McFarland	380	12	2
Ash	503	55	2
Cheyne	44	4	0
Share (as quoted in Smith)	119	15	1
Harvey, Dawson & Innes (as quoted in Hellwig)	230	21	2
Hellwig	67	12	0
	1629=90.6%	158=8.8%	10=0.6%

From this one can see that salivary gland tumors of the submaxillary gland are not as infrequent as one might expect.

Calculi are very frequently found in glandular tumors due to stasis of secretions and it might be expected that if a salivary gland tumor should be found with calculi it would be due to this cause.

The following two cases of mixed tumor of the submaxillary gland, both of which were diagnosed as enlargement of the gland due to obstruction by calculi, are presented.

#### REPORT OF CASES

CASE 1.—D. C. B., a 25-year-old white male was admitted to the Veterans Administration Hospital of Lebanon, Pennsylvania, on September 9, 1948, for a tumor in the right submandibular region. He gave a history of having noticed a lump on the right side of his neck about four years prior to admission. The mass remained the same size as when he first discovered it and one week before admission became painful.

Examination showed a firm mass about the size of an apricot just anterior to the sternocleidomastoid muscle on the right along

the margin of the mandible. The mass was free and nonadherent to skin or underlying tissue. No stone was palpable through the mouth or in the neck but x-ray film showed an irregularly dense shadow approximately 4 x 6 mm just inferior to the angle of the mandible in the region of the submaxillary gland which was diagnosed as a right submaxillary calculus. On September 20, 1948, the right submaxillary gland was excised, and found to be enlarged, firm and containing a small calculus. Pathological report described it as a submaxillary gland and attached mass with the gland tissue measuring 2.5 cm in all diameters, being normal in color and lobulation. It partially enclosed an encapsulated, firm mass which measured 3.2 x 2.2 x 2 cm. This mass was granular and mucoid and peeled out readily from adjacent gland. At one margin there was a calcific nodule within its capsule. Sections showed fairly normal submaxillary gland in one portion. In others epithelium resembling that of the basal layer of the skin was seen. In others it had a columnar arrangement. In other areas there were solid masses of epithelium, in still others the epithelium surrounded small spaces. Mixed with the epithelial tissue were large areas of myxoid fibrous tissue with small star shaped cells. Other areas of fibrous tissue were more solid and more cellular. One section showed a considerable amount of atypical cartilaginous tissue with numerous cartilage cells irregularly distributed. In the same section was a small mass of cancellous bone and osteoid tissue with numerous osteoblasts closely applied. This tissue was well differentiated.

Diagnosis was made of submaxillary gland and mixed tumor of submaxillary gland by Dr. S. S. Forrester, consultant in pathology. The patient made an uneventful recovery from operation and was discharged on September 28, 1949, and was advised to return in six months for re-check but did not return.

CASE 2.—J. T. A., a 53-year-old white male noticed a swelling under the left side of the jaw about five years prior to admission to the Veterans Administration Hospital, Lebanon, Pennsylvania, on August 16, 1949. About one and one-half years prior to his admission the swelling increased and was painful. He was operated on at a hospital where two stones were removed through his mouth. The mass became swollen and inflamed about six or seven times in the past year and a half and drained off and on. It had opened into the mouth on previous occasions about three times. Examination showed a firm mass about the size of an egg on the left side of the neck below the margin of the left mandible with a shallow sinus at the height of the mass. X-ray films showed a small calcific density on the left side of the neck about 2 mm in diameter in the

region of the submaxillary gland. No stones were palpable in the mouth. On August 22, 1949, the left submaxillary gland was dissected out and was found to be firmly adherent to a very much thickened and fibrotic skin and subcutaneous tissue. On section it showed a small calculus about 2 mm in diameter.

The removed mass was described by Dr. R. G. Gichner as being 4 x 2.5 x 2 cm, firm in texture. It had a definite fibrous capsule and the cut surface showed a tightly packed lobulated structure. The lobules showed a yellowish nuance and were separated by a translucent gray-white stroma. Associated with the mass was a calculus 0.5 cm in greatest diameter. Microscopic section showed scattered masses of cellular epithelial tissue, the cells sometimes arranged to form acini or duct like structures, surrounded by a dense fibrous tissue in some areas of which mucoid change was seen. There was much lymphosis throughout the tissue, although no typical germinal centers could be recognized. Individual cells showed none of the criteria of malignancy. Dr. Gichner gave as his impression: "Pleomorphic adenoma of salivary gland (mixed tumor) with calculus formation." The patient returned for a dental check-up on October 15, 1949, at which time there was a well healed scar, no drainage, and no indication of recurrence.

#### SUMMARY

Two cases of enlargement of the submaxillary glands have been presented with calculi in the gland which were proven to be salivary gland tumors. It is felt that inasmuch as stasis of secretions in a tumorous gland will cause calculi one must always consider the possibility of a salivary gland tumor even after the diagnosis of calculus of the submaxillary gland has been made by x-ray examination and stones have been removed.

1820 WEST THIRD STREET.

#### REFERENCES

1. Cheyne, V. D., Tiecke, R. W., and Horne, E. V.: A Review of So-called Mixed Tumors of the Salivary Glands Including an Analysis of Fifty Additional Cases, *Oral Surg., Oral Med. and Oral Path.* 1:359-402 (Apr.) 1948.
2. Hellwig, C. A.: Mixed Tumors of the Salivary Glands, *Arch. Path.* 40:1-10 (July) 1945.
3. Ahlbom, H. E.: Mucous and Salivary Gland Tumors. In Pack, George T., and Livingston, E. M.: *Treatment of Cancer and Allied Diseases*, New York, Paul B. Hoeber, Inc., Vol. 1, 1940, pp. 479-493.
4. McFarland, J.: Ninety Tumors of the Parotid Region, *Am. J. M. Sc.* 172:804, 1926; also *Surg., Gyn. and Obst.* 57:104, 1933; 63:457, 1936; 76:23, 34, 1943.
5. Ash, J. E.: Mixed Tumors of the Salivary Gland Type, *Am. J. Orthodon. and Oral Surg.* 33:522, 1947.
6. Smith, M. K.: Mixed Tumors of the Sublingual Gland, *Ann. Surg.* 109: 551-554 (Apr.) 1939.

## XI

### CLINICAL ELECTRONYSTAGMOGRAPHY

ARAM GLORIG, M.D.

SILVER SPRING, Md.

AND

ALEXANDER MAURO\*

NEW HAVEN, CONN.

During the past two years, an objective technique for the recording of nystagmus was developed in connection with the assessment of vestibular damage associated with streptomycin therapy. The complete methodology for clinical application will be described, namely, the technique for caloric stimulation and the electrical recording of eyeball movements.

#### CALORIC STIMULATION

To effect caloric stimulation of the vestibular system, the technique of Cawthorne, Hallpike and Fitzgerald<sup>2</sup> was chosen and modified<sup>3</sup> to insure a more constant minimal stimulus.

Briefly, the method consists of the following:

A thermos jug, acting as a reservoir, supplies water through a 30-in. tube with a 3-mm tip to the external auditory canal. A bronchoscopic specimen collecting tube, containing a centigrade thermometer, is fitted in the tubing, about 6 in. from the ear, to provide a careful check on the temperature of water flowing to the ear. Such an arrangement is necessary to check against any changes of temperature occurring in the reservoir or tubing. The temperature of the water may be 7° C. above or below normal body temperature (37° C.).

To obtain the most efficient stimulation of the fluid in the horizontal canal, the patient is placed in a supine position by means of a back-rest adjusted to an angle of 30° upward from the horizontal. Thus, the horizontal canal is oriented perpendicular to the ground. Irrigation of the external canal is carried out for 40 seconds via the tubing whose tip is moved frequently in and out to minimize

---

\*U.S.P.H. Junior Fellow, Division of Biophysics, Section of Neuro-Anatomy, Yale University School of Medicine.

any blocking of water flow by air bubbles. The overflow may be conveniently collected by means of an emesis basin placed beneath the patient's ear.

#### ELECTRICAL RECORDING

*Introduction.* Since the classic observations of Dewar and McKendrick<sup>1</sup> and of Holmgren<sup>4</sup> established the presence of a relatively invariant corneal-retinal potential difference, many investigators have continued critical studies to elucidate the character and origin of this potential. All information gathered supports the conclusion that the resting potential is a manifestation of basal physiological activity in the retinal cell mass. Various stimuli can perturb this resting potential, the most familiar being light, which gives rise to the electroretinogram.

By direct measurements on the excised bulbus, it has been found that the cornea is electrically the most positive, and the retina the most negative region. By observing the potential in situ, employing the internal and external canthi as the sites of the electrodes, Miles<sup>7</sup> has shown that the potential varies as the sine of the horizontal angle with respect to the primary line of regard. Thus, if the potential is measured between both external canthi, the magnitude is approximately summated for both eyes and will vary in magnitude and polarity as both eyes move in unison in either direction. The potential, as recorded, is, therefore, a measure of eye movement. Such a measurement for recording nystagmus was first tried by Schott<sup>8</sup> and later by Jung.<sup>5</sup> The waveform associated with nystagmoid movement is a typical "sawtooth" pattern (see Fig. 2). Most recently, an extended clinical paper has been published by Jung and Tönnies.<sup>6</sup>

*Recording Apparatus.* It was decided that the portable EKG machine (either battery or A.C. powered) would be most convenient as a recording device. Since the potential variation associated with nystagmus was of the order of 50 to 100 microvolts, the problem arose of building a "booster" or preamplifier of suitable characteristics:

- 1.) The preamplifier should be powered by batteries to minimize A.C. pickup problems and to make its application as flexible as possible.
- 2.) The circuit should have differential properties to minimize stray 60-cycle pickup from A.C. mains.
- 3.) Since the average commercial EKG machine is susceptible to overload and blocking, the preamplifier should possess a limiter characteristic so that it cannot deliver more than about 10 millivolts to the recording EKG machine. A circuit design was developed to satisfy all these requirements.

The circuit, as shown in Fig. 1, consists of a conventional differential input employing 1620 tubes to obtain low noise and low microphonics. The output stages consist of two resistor-capacitor coupled pentode stages employing 6SJ7 tubes. The limiter characteristic is attained by using two 1N34 germanium rectifiers arranged to clip the output for either positive or negative potential variations. Each rectifier is biased at 0.8 volt so that it begins to conduct quite effectively when the applied signal exceeds 1 volt. For example, consider the first rectifier; its anode is "biased" at -0.8 volt. Thus, if the output signal (across the 150-K resistor coupled via the 1-mfd. cap.) attains a value of one volt in the negative direction, the diode will conduct to limit the output at 1 volt. The second diode functions in a like manner for positive variations. Note the 500-K potentiometer in the grid circuit of the first stage is set by a screwdriver adjustment to give an output of 1 volt for 100 microvolts input. By means of the divider network—500-K and the 10-K potentiometer—a variable output signal to the EKG machine can be obtained up to a maximum of 20 millivolts.

The circuit design includes a calibrating network which can deliver 10, 20, 50 and 100 microvolts. The RUN-CALIBRATE switch (three-section single-pole double throw) serves to connect the 1620-stage either to the patient or to the calibrating network. The unit is powered by three 3-volt Willard miniature cells (BB-206-U) and a socket allows a connection for a trickle charger for periodic charging. The B battery supply is furnished by two Burgess 5308 batteries. All battery power is controlled by the main switch marked OFF—STAND BY—RUN.

A cable and terminal strip assembly is employed to connect the preamplifier to the conventional input leads of the EKG machine. The cable is the two-wire shielded type fitted on one end with a plug which inserts in the female receptacle at the output of the preamplifier and at the other end connects to the terminal strip, as indicated in Fig. 1. The posts on the terminal strip are marked C, LL, RL, LA, and RA respectively. Note the chassis "ground" of the preamplifier connects to RA and RL. Since the RL cable connects to the chassis "ground" of the EKG machine, a consistent "ground" connection is established throughout. The variable arm of the 10-K amplitude control connects to LA which is, therefore, the "active" lead. The posts marked C and LL merely serve as "dummy" posts to anchor leads C and LL.

Thus, by setting the selector switch in the EKG machine to the first position of operation, namely, Lead I, the system is ready to record signals. Note the EKG machine should be allowed to warm

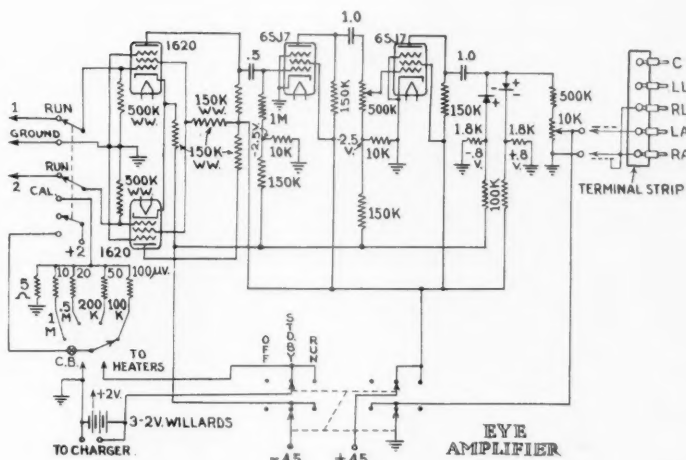


Fig. 1.—Circuit diagram of preamplifier.

up for several minutes. The preamplifier is allowed to warm up with the main switch in the STAND BY position and then switched to RUN for recording. Several calibration deflections should be recorded for input signals of 50 to 100 microvolts, in order to observe whether or not the entire system is in order.

**Electrode Placement.** As shown in Fig. 1, the two active eye electrodes 1 and 2, and ground electrode, are connected to the pre-amplifier input by a two-wire *shielded* cable. The cable is terminated at the preamplifier end by a suitable male connector and at the other end by a three-terminal strip to which can be attached the ground and the two active electrodes from the patient.

The electrodes employed are the type used in electroencephalogram applications, although silver discs, several millimeters in diameter, with flexible wires approximately 12 in. long, are equally suitable. The two active electrodes are moistened with electrode jelly and placed on opposite, external canthi, fixed in place by strips of cellulose tape; one over the electrode and another one-half inch posteriorly over the wire lead, in order to anchor it. In like manner, the ground or "indifferent" electrode is placed on the forehead. Care should be taken in checking the electrodes to be certain that they are making good contact. It is absolutely necessary that the ground of the system, i.e., *chassis of EKG machine*, be connected by a clip lead to the conduit ground or an easily accessible water pipe.

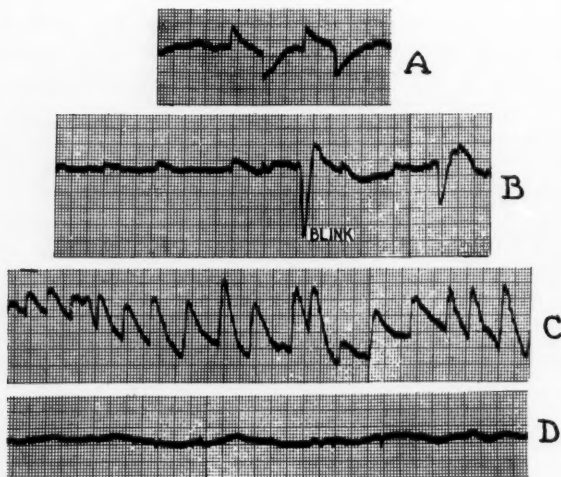


Fig. 2.—A. Recording of potential associated with a voluntary eye movement of  $4^{\circ}$ . B. Beginning of nystagmus as evidenced by sawtooth waveform of low amplitude. The recording associated with blinking is included to show marked difference. C. Nystagmus has developed maximally about 60 seconds later. Note typical sawtooth waveform is very striking. D. Endpoint about 110 seconds. Base line is substantially flat.

Failure to observe these points will almost invariably cause excessive 60-cycle pickup to mar the recording.

#### GENERAL NOTES OF PRECAUTION

The number of voluntary eye movements will tend to be minimized if the patient is made as comfortable as possible. In addition to arranging conditions to insure comfort, a few words of explanation concerning the test will help to relax the irritable patient. The absence of any direct sources of illumination in the visual field is to be recommended.

The most important factor serving to minimize voluntary eye movements is the provision of a designated spot or small lamp situated 10-15 ft. away from the patient. A gentle reminder during the test procedure to "keep looking at the spot" will maintain the patient's fixation very effectively along the primary line of regard.

The problem of extraneous interference, such as 60-cycle pickup from the power lines, will not arise if care is taken in providing a good ground connection and in placing the electrodes on the subject with care. A good ground is absolutely imperative. Poor

electrode placement usually involves inadequate preconditioning of the skin with electrode jelly. It is wise to record for several seconds before irrigating the subject's auditory canal to check if any interference is being recorded.

#### DISCUSSION

A technique for the recording of nystagmus is described for use by clinicians. The basic recording instrument is the standard electrocardiograph supplemented by a "booster" or preamplifier. The entire system is easily portable so as to make it useful anywhere in the clinical service. The marked advantage of the objective recording is primarily the establishment of a permanent recording to permit the detailed study of such parameters as waveform, frequency, and time duration of nystagmus activity of various patients.

The authors are very grateful to Mr. B. C. Bruestle for his kindness in helping with the preparation of the manuscript.

The preamplifier described in this paper, is now available from Bioelectronics Laboratory, 16 Dow Street, New Haven, Connecticut.

200 EAST INDIAN SPRING DRIVE.

#### REFERENCES

1. Dewar, J., and McKendrick, J. G.: On the Physiological Action of Light, *Trans. Roy. Soc. Edinburgh* 27:1876, 1941.
2. Fitzgerald, G., Cawthorne, T. E., and Hallpike, C. S.: Studies in Human Vestibular Function: I. Observations on the Directional Preponderance of Caloric Nystagmus Resulting from Cerebral Lesions, *Brain* 65:1115, 1942.
3. Glogig, Aram, and Fowler, E. P.: Tests for Labyrinth Function Following Streptomycin Therapy, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 56:379 (June) 1947.
4. Holmgren, F. A.: Method att objektivera jusintryck pa retina, *Upsala läkaref förh.* 1:177, 1865-1866.
5. Jung, R.: Eine Elektrische Methode zur Mehrfachen Registrierung von Augenbewegungen und Nystagmus, *Klin. Wschr.* 1:21, 1939.
6. Jung, R., and Tönnies, J. F.: Die Registrierung und Auswertung der Drehnystagmus Beim Menschen, *Klin. Wschr.* 1:513-521 (Sept.) 1948.
7. Miles, W. R.: The Steady Polarity Potential of the Human Eye, *Proc. Nat. Acad. Sc.* 25:25, 1939.
8. Schott, E.: Über die Registrierung des Nystagmus und anderer Augenbewegungen vermittels des Saitengalvanometers, *Deut. Arch. f. Klin. Med.* 140:79, 1922.

## XII

### NEURALGIC SYNDROMES ASSOCIATED WITH APLASIA OF THE FRONTAL SINUS

ETTORE GIUFFRIDA, M.D.

CATANIA, ITALY

I wish to call attention to a syndrome which is characterized by frontal headache, more or less continuous, with paroxysms and short periods of remission, lasting several hours. Rhinologic examination is negative. There is no trace of exudation and only occasionally a slight hypertrophy of the middle turbinate. X-ray examination shows one or both sinuses to be absent. Sometimes a sinus is replaced by an ethmoidal cell which seems not to have penetrated between the two laminae of the frontal bone.

Sometimes in the place of the frontal sinus there is an ossified thickening which seems to be an obliteration of a previously developed sinus.

Herbert Tilley<sup>5</sup> for the first time called attention to this syndrome before the Royal Society of Medicine in London in 1928. He reported the case of a 29-year-old woman who, every four or five weeks, had cephalalgic attacks which lasted for eight to ten days. X-ray examination showed the frontal sinus completely absent. Tilley operated upon the frontal bone in the sinus region, after which the headaches abated.

About in the same period (1928) also Cambrelin<sup>1</sup> described two similar cases, under the designation of "ossifying frontal sinusitis." He attributed the obliteration of the sinus and the headaches to a chronic ossifying inflammation. In 1936 Skillern also described "obliterating frontal sinusitis." According to the author the process was caused by a low grade hyperplastic inflammation of slow development occasioned by attenuated germs. The process is characterized by a slow forming of granulations and by a periosteal involvement with thickening osteitis, which causes the obliteration of the sinus. Trauma should have, according to Skillern,<sup>10</sup> a great importance in the genesis of the process, which is characterized by headache, toxemia, nervousness and gastro-enteric disturbances. Ac-

---

From the Department of Otorhinolaryngology of the Vittorio Emanuele Hospital, Catania, Italy.

cording to Skillern, the roentgen interpretation is important in the diagnosis of aplasia of the sinus, a contra-indication to surgery. On the contrary, the treatment must be only surgical and consist in the radical operation of the frontal sinus.

In 1940 also Gignoux<sup>3</sup> pointed out this neuralgic syndrome associated with absence of the frontal sinus. This author gave an interpretation similar to that of Skillern, but he did not exclude the possibility of a reflex neuralgia.

#### REPORT OF CASES

CASE 1.—A young man aged 20 had a history of having been struck with a stone in his left frontal region. He began to suffer from headache on the left side with changes in atmospheric conditions. Examination of nasal fossae was negative. Radiography demonstrated that the frontal sinus was wanting. Following the idea of an obliterating frontal sinusitis, I operated externally on the left frontal region and found a thick bone in the place of the sinus; the nasofrontal duct was entirely absent. When I removed the thick bone, I penetrated the right frontal sinus. The wound recovered per primam. Headaches disappeared for some time, but afterwards they appeared again occasionally and weakly. An anti-allergic treatment was then performed with magnesium sulfate intravenously and recovery was obtained.

CASE 2.—A woman aged 36 suffered from headache for two years. She had had antineuralgic treatment without result. There was hypertrophy of the turbinates. X-ray examination showed the sinuses to be rudimentary with thickening of the walls. Inferior and middle turbinotomy and general anti-allergic treatment brought recovery.

CASE 3.—A young man aged 20 had received a blow on the head by an ox, five years before, in the right frontal region. Since then, he suffered always from frontal headache, first occasionally, and recently almost continuously. Radiography showed an obliteration of both frontal sinuses, with hyperostosis in the right side. Also in this case turbinotomy and ethmoidotomy, followed by anti-allergic treatment obtained relief from the headache.

CASES 4, 5 and 6 concern, respectively, a woman aged 64, a young woman aged 31 and one aged 27. In these cases frontal sinuses are wanting. Recovery from the neuralgic syndrome was obtained by simple anti-allergic treatment (magnesium sulfate intravenously, gynengen).

In the interpretation of this symptom complex there are two fundamental points:

1. The peculiar nature of the process: aplasia or obliteration originating in frontal osteitis.
2. Connections between neuralgia and the sinus process: is it a neuralgia caused by a sinusitis or is it independent from it?

As to the nature of the process, I believe that osteogenic hyperplastic inflammation is always the cause. The evolution of this process differs only in relation to the age of the patient when the inflammation appears. In childhood the process causes an aplasia of the sinus. The inhibiting influence of trauma in ethmoidofrontal pneumatization is already well known. Trauma though slight may reduce the penetrating power of ethmoidal mucosa with an agenesis of the sinus.

In other cases, I think that an osteoproliferative inflammation in a sinus already developed (Cambrelin,<sup>1</sup> Skillern<sup>10</sup>) can better explain sinus obliteration.

The clinical course and the results of the treatment in my cases do not allow us to consider neuralgia as a result of sinus inflammation. In these six cases headache disappeared in consequence of the anti-allergic treatment and contrary to Skillern's opinion,<sup>10</sup> recovery occurred following operation on the sinus and nasal cavities too, as well as in the cases in which there was no surgery.

I think, therefore, that the syndrome presents the character of an allergic phenomenon together with sinus inflammation.

Pietrantonio's observations<sup>7</sup> and those of Nuhmann, Urbach and Hansel indicate an allergic element in the hyperplastic sinus inflammations earlier studied by Sluder, Dutheillet de la Mothe and Sourdille.<sup>2</sup> I had described in 1937, among these hyperplastic forms, also those with osteoproliferative obliteration. The allergic phenomenon enables us also to explain the neuralgic manifestation.

It is necessary to admit that the neuralgic syndrome which accompanies the absence of one or both frontal sinuses, is to be referred to the constitution of the patient, and rightly to a trigemino-sympathetic weakness, or to an amphohypertonía.

The fronto-ethmoidal process may be considered responsible for the headache only reflexly.

The failure of surgical treatment in these cases is another demonstration of the necessity to modify the constitutional reactivity of the patient.<sup>11</sup>

VIA SALVATORE PAOLA 15.

## REFERENCES

1. Cambrelin, G.: A propos de deux cas de sinusite frontale ossifiante, *Rev. de Laryngologie* 49:130 (Feb.) 1928.
2. Dutheillet de la Mothe, G., and Sourdille, M.: Les cephalalgies frontales rhinogenes et leur traitement chirurgical, *Rapp. Soc. Franç. O. R. L.* 42:155, 1929.
3. Gignoux, M. A.: Troubles causées par l'absence unilaterale de sinus frontal, *Les Ann. de Laryngologie* 60:213 (Apr.) 1940.
4. Giuffrida, E.: Patologia e clinica dei seni frontali, *Relazione Soc. Ital. O. R. L.* 33:68, 1937.
5. Tilley, Herbert: Très petit sinus frontal associé à des cephalées graves. Absence de suppuration, *Royal Soc. Med. London*, May 31, 1928. Ref. in *Rev. de Laryngologie* 49:361, 1928.
6. McLaurin, J.: Headaches of Otorhinologic Origin, *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 50:469 (June) 1941.
7. Pietrantoni, L.: Nevralgie trigemino-simpatiche da sinusiti etmoido-mascellari iperplastiche, *Atti Congr. Ital. O. N. O.* 2:137, 1931.
8. Rethi, A.: Nevrites trigemellaires d'origine rhinogène, *Les Ann. de Laryngologie* 64:431, 1947.
9. Sargnon, A.: Nez et oeil, *Les Arch. Hospitalieres*, Paris, 1936, p. 136.
10. Skillern, M.: Sinusite frontale oblitérante. Ref. in *Il Valsalva* 12:267, 1936.
11. Worms, J.: Cefalee e disturbi oculari di origine nasale, *Riv. Oto-neuro-oftalmologica* 4:496, 1925.

### XIII

## BACTERIOLOGIC AND CLINICAL INTERPRETATION OF THE FLORA OF THE NOSE AND NASOPHARYNX IN ADULTS

JOSEPH L. GOLDMAN, M.D.

NEW YORK, N. Y.

In order to re-evaluate the significance of the nose, sinuses and nasopharynx as foci of infection in relation to systemic diseases, I have undertaken bacteriologic and cytologic studies of these sites in health and disease. In this paper, I am reporting on studies made to ascertain what is the normal flora of the nose and nasopharynx and what is the relationship of these flora to each other. An evaluation of the flora of the normal nose and nasopharynx is essential before one can interpret the clinical significance of the micro-organisms found in infections.

There have been changes of attitude among internists and others in the past decade or two toward the relationship of so-called foci of infection of the upper respiratory tract to systemic diseases. The tendency has been to place less reliance on such relationships, particularly when the question of removing the focus surgically has arisen. Yet, it has been observed not infrequently that eye infections, arthritic manifestations, asthmatic breathing, evidences of vascular allergy or other types of systemic reactions have subsided with the cure or control of a sinus infection. It is probable that much of the skepticism regarding these relationships has resulted from disappointments and failures in the management of these conditions. A lack of definite information concerning significant bacteriologic findings of the upper respiratory tract may have been responsible for some of these failures.

Rhinologists have relied mainly on the finding of pus or secretion in the nose or in the sinuses as the criterion of infection. With the advances that are being made by bacteriologic and cytologic studies of the nose and sinuses, in health and in disease, it is becoming clear that the mere presence of secretion in the nose or sinuses cannot be accepted as the sole diagnostic determinant of a focus of infection. Examination of secretion from the nose or a maxillary antrum, even when obtained in large amounts, may reveal only cellular elements

---

From the Otolaryngological Service and Division of Bacteriology of The Mount Sinai Hospital, New York, N. Y.

TABLE 1.  
FLORA OF NORMAL NOSE AND NASOPHARYNX  
35 CASES—MOUNT SINAI HOSPITAL  
WINTER 1941-1942 AND SPRING 1942

	RIGHT	LEFT	NASOPHARYNX
<i>Pneumococcus</i>	0	1 (13)*	2 (1-13)
<i>Streptococcus hemolyticus</i>	0	0	12
<i>Streptococcus viridans</i>	2	1	18
<i>Streptococcus non-hemolyticus</i>	0	0	1
<i>Staphylococcus aureus</i>	6	6	4
A	(5)	(5)	(4)
B	(1)	(1)	(0)
<i>Staphylococcus albus</i>	31	34	10
A	(2)	(3)	(1)
B	(29)	(31)	(9)
Diphtheroids	5	5	2
<i>Neisseria</i> species	0	0	22
No growth	2	1	2

\**Pneumococcus* also in nasopharynx in this case.

devoid of bacteria, with eosinophils ranging up to 100%. Cultures of such secretion may confirm the absence of bacteria or demonstrate micro-organisms which are nonpathogenic in character and which, from the clinical and immunological point of view, generally have no significance. On the other hand, culture of a congested or edematous mucous membrane of the nose, without the presence of secretion, as well as a scant amount of secretion from a sinus, can yield many clinically significant pathogenic micro-organisms.

Thus, in order to evaluate the findings in nasal and sinus infections accurately, one must be able to interpret the character, number and pathogenicity of the prevailing micro-organisms. In other words, it is desirable that the study of smears of nasal and sinus secretion in regard to the cellular elements and number of bacteria present and the culture of the secretion become a routine procedure in rhinologic examinations, particularly for the accurate appraisal of foci of infection. It is believed that in this way the specificity of infections in the nose, sinuses and nasopharynx in relation to various systemic diseases will become clarified. This approach also will afford

TABLE 2.  
FLORA OF NORMAL NOSE AND NASOPHARYNX  
31 CASES—REGIONAL HOSPITAL, DREW FIELD, FLORIDA  
WINTER-SPRING 1944

	RIGHT	LEFT	NASOPHARYNX
<i>Pneumococcus</i>	1*	0	17
<i>Streptococcus hemolyticus</i>	0	0	0
<i>Streptococcus viridans</i>	0	0	12
<i>Streptococcus non-hemolyticus</i>	10	9	9
<i>Staphylococcus aureus</i>	21	22	27
A	(3)	(1)	(12)
B	(18)	(21)	(15)
<i>Staphylococcus albus</i>	9	10	7
A	(1)	(1)	(3)
B	(8)	(9)	(4)
Diphtheroids	4	4	10
<i>Neisseria</i> species	1	0	7
Others	2	1	2
No growth	3	1	0

\**Pneumococcus* also in nasopharynx in this case.

the chance to determine more precisely the indications for the proper use of antibiotic therapy.

#### TECHNIC

The technic used to obtain nasal and nasopharyngeal cultures in these studies is as follows: A platinum loop is used for both cultures. For nasopharyngeal cultures a larger loop is preferable. In taking nasal cultures, the platinum loop is passed into the sphenothmoidal recess and middle meatus and over the turbinates. It is believed that the platinum loop is far superior to a cotton applicator, which is liable to cause gagging and contamination from nasopharyngeal secretion. The nasopharyngeal culture was made by passing a curved loop through the mouth into the nasopharynx. When performed by a laryngologist, using a head mirror, contamination usually can be avoided. Cultures are made routinely on blood agar plates alone. Not only are micro-organisms easily grown and identified on blood agar, but the opportunity for quantitative estimation is also afforded by this medium and method.

TABLE 3.  
FLORA OF NORMAL NOSE AND NASOPHARYNX  
72 CASES—WILLIAM BEAUMONT GENERAL HOSPITAL, EL PASO, TEXAS  
DECEMBER 1945-JUNE 1946

	RIGHT	LEFT	NASOPHARYNX
<i>Pneumococcus</i>	0	0	8
<i>Streptococcus hemolyticus</i>	1*	0	13
<i>Streptococcus viridans</i>	1	2	14
<i>Streptococcus non-hemolyticus</i>	2	2	32
<i>Staphylococcus aureus</i>	23 (2)	29 (4)	44 (6)
m+ (A)	(6)	(6)	(8)
m- (B)	(15)	(19)	(30)
<i>Staphylococcus albus</i>	19 (3)	22 (4)	16 (3)
m+ (A)	(3)	(2)	(3)
m- (B)	(13)	(16)	(10)
Diphtheroids	0	0	1
<i>Neisseria</i> species	4	2	28
No growth	28	22	1

\*Many hemolytic streptococci in nasopharynx in this case.

All pneumococci in December and January. All hemolytic streptococci in April and May.

A discussion is appropriate at this point concerning methods being used to determine the pathogenicity of the staphylococcus. This micro-organism is the one most commonly found in the nose both in disease and in health. There appears to be no sharp differentiation between the pathogenic and nonpathogenic staphylococcus from any clinical aspect. To establish such differentiation, considerable investigation<sup>1, 2</sup> has been conducted recently, using various biochemical tests. According to almost all investigators, the most reliable test indicative of pathogenicity is the coagulation of plasma. Of interest to us is that Hallman<sup>3</sup> found that from 40-60% of staphylococci from the normal nose were potential pathogens, as shown by the presence of coagulase, and that these results have been corroborated by McFarlan.<sup>4</sup> The fermentation of mannite is considered next in worth in denoting staphylococci as pathogens and as a valuable confirmatory test to the plasma coagulation reaction. It is further believed that the properties of hemolysis and pigment formation, when considered alone, are much less reliable in labelling staphylococci as pathogens. In general, the coagulation of plasma and fermentation of mannite by a hemolytic staphylococcus are re-

TABLE 4.  
FLORA OF NORMAL NOSE AND NASOPHARYNX  
8 CASES—NEW YORK  
1946-1947

	RIGHT	LEFT	NASOPHARYNX
<i>Pneumococcus</i>	0	0	5
<i>Streptococcus hemolyticus</i>	0	0	2
<i>Streptococcus viridans</i>	0	0	7
<i>Streptococcus non-hemolyticus</i>	0	0	1
<i>Staphylococcus aureus</i>	1	2	1
A	(0)	(0)	(0)
B	(1)	(2)	(1)
<i>Staphylococcus albus</i>	3	3	2
A	(0)	(0)	(0)
B	(3)	(3)	(2)
Diphtheroids	2	2	5
<i>Neisseria</i> species	0	1	5
Friedlander's bacillus	0	0	1
Others	3	2	0

garded by most authorities as highly indicative that one is dealing with a pathogenic or potentially pathogenic micro-organism. These reactions, occurring together, have been found pathogenically valid in over 90% of a large series of cases.<sup>1, 3, 5</sup> Staphylococci whose tests indicate pathogenicity are designated as A, those whose tests indicate nonpathogenicity as B.

#### MATERIAL

First, an analysis of the bacterial flora of noses and nasopharynges of individuals with no evidence of nasal or sinus disease will be presented. This status was determined by history, examination and transillumination of the sinuses. These findings represent studies made in different parts of the country. Secondly, an analysis of the bacterial flora of the noses and nasopharynges of patients whose nasal condition was diagnosed as vasomotor rhinitis will be shown.

In Table 1 are presented 35 patients with normal nasal cavities and sinuses who were examined for foci of infection at The Mount Sinai Hospital during 1941-1942. Most of these patients were suffering from rheumatoid arthritis. In this group, the pneumococcus

TABLE 5.  
FLORA OF NORMAL NOSE AND NASOPHARYNX  
146 CASES

	RIGHT	LEFT	NASOPHARYNX
<i>Pneumococcus</i>	1*	1*	32 (21+%)
<i>Streptococcus hemolyticus</i>	1*	0	27 (18+%)
<i>Streptococcus viridans</i>	3	3	51
<i>Streptococcus non-hemolyticus</i>	12	11	43
<i>Staphylococcus aureus</i>	51	59	76
A	(14)	(12)	(24)
B	(35)	(43)	(46)
<i>Staphylococcus albus</i>	62	69	35
A	(6)	(6)	(7)
B	(53)	(59)	(25)
Diphtheroids	11	11	18
<i>Neisseria</i> species	5	3	62
Friedlander's bacillus	0	0	1
Others	5	3	2
No growth	33	24	3

\*Growth in nasopharynx in same case.

was isolated from the left nasal cavity of only one patient and it should be noted that the same pneumococcus was also present in the nasopharynx of this patient. The pneumococcus was obtained from the nasopharynx in 2 patients. Of significance in this series is the fact that *Streptococcus hemolyticus* was not obtained at all on culturing the nasal cavities, but was isolated from the nasopharynx in 12 patients. *Streptococcus viridans* was found in the right nasal cavity twice, left nasal cavity once, yet in the nasopharynx of 18 patients. One is impressed with the high incidence of *Staphylococcus albus* B in these normal nasal cavities.

In Table 2 are reported the findings in 31 enlisted men examined at a military hospital in Florida during the winter and spring of 1944. These soldiers also had normal nasal cavities and sinuses. Again the pneumococcus was isolated from one nasal cavity in an individual who had many pneumococci in the nasopharynx. On the other hand, the pneumococcus was isolated from the nasopharynx in 17 persons. It is striking that in this group *Streptococcus hemolyticus* was lacking from all cultures. *Streptococcus viridans* was

TABLE 6.  
FLORA OF NOSE AND NASOPHARYNX  
37 CASES OF VASOMOTOR RHINITIS  
1946-1947

	RIGHT	LEFT	NASOPHARYNX
<i>Pneumococcus</i>	2*	1*	21 (57%)
<i>Streptococcus hemolyticus</i>	0	0	5 (13+%)
<i>Streptococcus viridans</i>	0	2	18
<i>Streptococcus non-hemolyticus</i>	0	0	1
<i>Staphylococcus aureus</i>	10	10	7
A	(6)	(4)	(4)
B	(4)	(6)	(3)
<i>Staphylococcus albus</i>	5	8	2
A	(0)	(0)	(1)
B	(5)	(8)	(1)
Diphtheroids	6	6	10
<i>Neisseria</i> species	6	6	12
Friedlander's bacillus	0	0	1
<i>H. parainfluenzae</i>	2	2	2

\**Pneumococcus* also in nasopharynx in these cases.

not obtained from the nose in anyone, but from the nasopharynx in 12 individuals. In this series, *Staphylococcus aureus* and *albus* B were the predominant micro-organisms in the nose.

In Table 3 are listed the results on culturing the nasal cavities and nasopharynges of 72 patients with normal upper respiratory tracts at William Beaumont General Hospital, El Paso, Texas, from December 1945 to June 1946. In this series the pneumococcus was not isolated from the nose in any patient and *Streptococcus hemolyticus* was obtained from one nasal cavity in an individual whose nasopharynx contained many hemolytic streptococci. The nasopharyngeal cultures, however, yielded pneumococci in 8 persons and hemolytic streptococci in 13. It is of interest that all the pneumococci were isolated in December and January and all the hemolytic streptococci in April and May. The staphylococci, particularly the nonpathogenic B variety, again prevailed in the nose.

In Table 4 are recorded 8 patients who were examined in my office in 1946-1947 for foci of infection and found to have normal

nasal cavities and sinuses. No pneumococci or streptococci were isolated from the nose. Cultures of the nasopharynx yielded the pneumococcus in 5 patients, *Streptococcus hemolyticus* in 2, *Streptococcus viridans* in 7.

The compilation of the above 146 normal noses and nasopharynges in Table 5 shows that the pneumococcus and *Streptococcus hemolyticus* were isolated only in 3 instances in individuals who carried these same micro-organisms in the nasopharynx. On the other hand, the pneumococcus was isolated from the nasopharynx in 32 persons (21+) and the *Streptococcus hemolyticus* in 27 (18+). *Streptococcus viridans* was isolated from both the right and left nasal cavities in 3 instances, while this micro-organism was obtained from the nasopharynx in 51 individuals. The incidence of staphylococci in the nose was high and again it should be noted that the nonpathogenic B variety was greatly preponderant. It should be mentioned that staphylococci when isolated from normal nasal cavities were, as a rule, sparse in number; also that the usual number of micro-organisms isolated from one nasal cavity was one or two, while several were commonly obtained from the nasopharynx.

The analysis presented in Table 6 is of special interest. The nasal condition of these 37 patients, examined in my office during 1946-1947, was diagnosed as vasomotor rhinitis. Their difficulties were due mainly to swollen, congested inferior turbinates and mucous membrane. The etiologic factors were largely physical, constitutional and emotional. With a few exceptions, the sinuses of these patients were proved to be free from infection by diagnostic lavage and culture. The bacteriologic findings in these patients were similar to those obtained in the patients with normal noses. The pneumococcus was isolated from the nasal cavities in 3 patients but in each instance this micro-organism was also present in the nasopharynx in large numbers, while the nasopharynx yielded the pneumococcus in 21 persons (57+). *Streptococcus hemolyticus* was not obtained from the nose of any patient, yet was isolated from the nasopharynx in 5 individuals (13+). *Streptococcus viridans* was obtained from the nose in 2 patients and from the nasopharynx in 18. The staphylococci obtained from the nasal cavities of these patients were usually few in number.

#### SUMMARY AND CONCLUSION

To summarize, staphylococcus of the nonpathogenic variety as determined by the biochemical methods mentioned in the text, was the most common micro-organism found in the normal noses in the entire series studied. This micro-organism was present in small and occasionally moderate numbers. It should be noted that the normal

nose often yielded a sterile culture. Diphtheroids and *Streptococcus non-hemolyticus* were isolated in a small percentage of the normal noses and *Streptococcus viridans* only in several cases. Usually only one or two of these micro-organisms were obtained from one nasal cavity.

In the normal nasopharynxes, pneumococcus was obtained in 21% of the persons studied, *Streptococcus hemolyticus* in 18%. *Streptococcus viridans*, *Streptococcus non-hemolyticus*, staphylococci and neisseria species were present in greater percentages. These micro-organisms were usually isolated in large numbers and several were frequently cultured from a single nasopharynx.

Pneumococci and hemolytic streptococci were not found in normal nasal cavities without also existing in the nasopharynx. In this series these micro-organisms were isolated simultaneously from the nasal cavities and nasopharynx only in several instances.

The bacterial flora of the noses and nasopharynxes of the patients whose nasal conditions were diagnosed as vasomotor rhinitis was similar to that found in normal noses and nasopharynxes.

Thus, in conclusion, it can be stated that for diagnostic purposes, the presence in the nose of pneumococci and hemolytic streptococci particularly and pure cultures of large numbers of *Streptococcus viridans*, *Streptococcus non-hemolyticus*, *Staphylococcus aureus* A, Friedländer's bacillus and *Hemophilus influenzae* is generally indicative of the existence of an infection. *Staphylococcus aureus* A may be an exception which requires further physical evidence of infection. It is possible that surface infections without involvement of the sinuses may be of greater clinical significance as foci of infections than hitherto assumed.

On the other hand, the micro-organisms which have been enumerated can be isolated from nasopharynxes which show no physical evidence of infection. It appears that the nasopharynx harbors or might be considered the carrier-focus of the pathogenic micro-organisms which can spread into the nose and sinuses when the defensive antibacterial barriers are broken by lowered tissue resistance or disease processes, as for instance, the common cold. An important question which must be considered in the problem of focal infection is whether the mere existence of pathogenic micro-organisms in the nasopharynx, particularly in large numbers, can act as an active or potential focus of infection. Another significant problem for solution is to determine where the status of carrier-focus ends and low-grade infection of the nasopharynx begins. The role that lymphoid tissue in the nasopharynx plays in making this area

a carrier-focus, and the local use of antibiotics and other antibacterial drugs to prevent the spread of nasopharyngeal bacteria into the nose, are pertinent collateral problems.

Finally, it seems important that our concept of a focus of infection in the upper respiratory tract should be defined more specifically in terms of bacteriologic agents. In this way a better appreciation and understanding between foci of infection and various systemic diseases will be created.

1050 PARK AVENUE.

#### REFERENCES

1. Chapman, G. H., Berens, C., Nilson, E. L., and Curcio, L. G.: The Differentiation of Pathogenic Staphylococci from Non-pathogenic Types, *J. Bact.* 35:311-334, 1938.
2. Blair, J. E.: The Pathogenic Staphylococci, *Bact. Rev.* 3:97-146, 1939. (Review and Bibliography)
3. Hallman, F. A.: Pathogenic Staphylococci in the Anterior Nares: Their Incidence and Differentiation, *Proc. Soc. Exp. Biol. and Med.* 36:789-794, 1937.
4. McFarlan, A. M.: Incidence of Pathogenic Staphylococci in the Nose, *Brit. M. J.* 2:939-941, 1938.
5. Cruikshank, R.: Staphylocoagulase, *J. Path. Bact.* 45:295-303, 1937.

## XIV

### THE CAROTID CANAL

#### AS A PATHWAY FOR EXTENSION OF INFECTION IN THE TEMPORAL BONE

J. G. DRUSS, M.D.

NEW YORK CITY, N. Y.

The close proximity of the carotid canal to the tympanic cavity, petrosa and other structures in the temporal bone makes it particularly susceptible to the infections which occur in all these structures, and because the carotid artery with its plexus of veins lodges there, it plays a significant role in the transmission of these infections to other parts of the body.<sup>1-3</sup> The carotid canal is situated in the anteromedial portion of the petrous pyramid and shows considerable variation with regard to size and shape.<sup>4,5</sup> It is comprised of a vertical (ascending) and a horizontal portion. It is very thin in places, particularly in its horizontal division; and here many dehiscences with the bone entirely lacking have been demonstrated histologically by Guild,<sup>6</sup> Wilson, Gaardsmoe and Anson,<sup>7</sup> and others. The canaliculus caroticotympanicus, through which passes the caroticotympanic artery can be seen near the external carotid foramen. It is also perforated at many sites by other fine blood vessels and nerves.<sup>2</sup>

The carotid canal is occupied chiefly by the internal carotid artery. This vessel is enveloped by the carotid plexus of veins and of nerves, and by lymphatics, all held together by a loose connective tissue sheath. These structures, inextricably woven into one fabric embrace the artery proper and probably serve to dampen its pulsations. It is for this reason that upon removal of the bony canal at operation and exposure of the contents within, the carotid artery is seldom seen to pulsate.

The carotid plexus of veins continues on medially as the cavernous sinus which it resembles very closely in appearance, and it empties

---

From the Otolaryngological Service of Dr. R. Kramer and the Department of Laboratories of the Mount Sinai Hospital, New York City, N. Y.

Read before the Section on Otolaryngology, the Academy of Medicine, December, 1948.

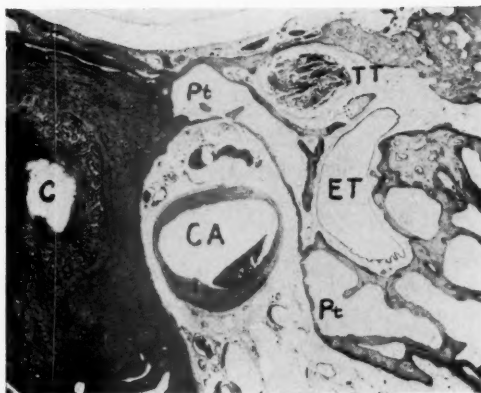


Fig. 1.—Photomicrograph of section through the carotid canal showing the close relationship between the eustachian tube and peritubal cells and the carotid canal. Note the dehiscences in the relatively thin bony canal wall. C represents cochlea; CA, carotid artery; ET, eustachian tube; Pt, peritubal cells; and TT, tensor tympani muscle.

into the jugular bulb below and behind. It receives venous tributaries from the adjacent structures including the tympanum, eustachian tube, peritubal cells, cochlea and the petrosa.<sup>8</sup>

Ferron<sup>9</sup> has shown that the carotid sheath continues into the cavernous portion and constitutes the deep layers of the wall of the cavernous sinus, while the superficial layer is formed by the dura mater.

The carotid plexus of nerves constitutes a part of the sympathetic nervous system and is derived chiefly from the ascending branch of the supracervical ganglion. Besides giving off filaments to the tympanic plexus it contributes branches to the deep petrosal nerve, the latter joining with the great superficial petrosal to form the vidian nerve. Other fibers of the carotid plexus are given off from the superior petrosal nerve to supply the ciliary muscle and thus control dilatation of the pupil. Still other fibers, according to Chorobski and Penfield<sup>10</sup> control the caliber of the terminal branches of the carotid artery in the cranium. Of all the branches just enumerated, those that pierce the carotid canal and consequently constitute potential pathways of infection, are of particular significance in this communication.

The lymphatics within the carotid canal, due to the collapsed state of their walls under normal conditions, are seldom discernible



Fig. 2.—Photomicrograph showing thrombosis of the pericarotid plexus of veins secondary to suppurative process in the eustachian tube. C represents cochlea; CP, carotid plexus; and ET, eustachian tube.

even under the microscope. In the presence of acute infections, however, their walls may become distended by the accumulation of inflammatory cells; they then can be recognized more readily and be identified as the perineural spaces which surround the individual nerves.

*Relationship with the Tympanic Cavity and the Eustachian Tube.* Although the carotid canal is in reality a part of the inner tympanic wall and although it is quite thin in young subjects and is perforated by the caroticotympanic vessels, the acute infections usually occurring in the tympanic cavity have seldom been seen to invade the canal and reach the structures within. Such extension has been observed with greater frequency in the more virulent infections, especially in those complicating the acute exanthemata and in tuberculous otitis.<sup>11-14</sup> In both of these conditions bone necrosis is not an unusual finding. Also chronic suppurations of the middle ear, particularly those with cholesteatoma, are more prone to invade the carotid canal. It is of interest to note that this route of extension was first described by Körner<sup>15</sup> over 50 years ago, and case reports illustrating such extension have subsequently appeared from time to time.<sup>16, 17</sup>

The eustachian tube lies in juxtaposition with the carotid canal for a short distance as they course through the petrosa. The bony canal wall separating these structures is, as previously stated, very thin in areas and even entirely lacking in others (Fig. 1). Thus there are many sites where the mucosa lining the eustachian tube lies in direct contact with the pericarotid tissues. This applies also to the adjacent peritubal pneumatic spaces and bone marrow. Close communication between the carotid contents and all these bony structures is also established by means of small vessels which arise in the submucosa or bone marrow, penetrate the bony canal wall and enter the carotid plexus. Notwithstanding the presence of the many potential avenues for the spread of infection, it is surprising that extension does not take place more frequently; particularly since it is well established that the submucosa of the eustachian tube and tympanum may harbor for a prolonged period a dormant infectious process which ultimately becomes activated and spreads to the adjacent structures. Cases in which an infection in the eustachian tube has spread to the carotid canal have been described by Schlender,<sup>18</sup> Jones,<sup>19</sup> Fowler,<sup>20</sup> and others. Figure 2 portrays a similar picture taken from a patient included in our study who had died from sepsis and meningitis secondary to a virulent otitis media. There can be no question that such extension would be observed more frequently if careful postmortem examination with histologic sectioning of the temporal bone was routinely performed in all cases of middle ear suppuration.

*Relationship with the Petrosa.* The variations in size of the carotid canal and the thickness of its bony wall are probably dependent upon the inherent anatomic structure of the petrosa itself. In the well pneumatized petrosa where the pneumatic spaces almost entirely surround the carotid canal it is the rule to find a very thin bony wall with many dehiscences (this has been well demonstrated by the histologic studies of Guild,<sup>6</sup> Jones<sup>19</sup> and others); in the diploic or sclerotic petrosa, on the other hand, the bony wall is usually much thicker and contains fewer dehiscences.

Suppurations of the petrous pyramid have not infrequently been observed, both clinically and histologically, to extend to the carotid canal. Though our material is insufficient to warrant making comparative studies, it would appear from the anatomic standpoint alone, that such extension takes place with greater facility in a well pneumatized petrosa than in a diploic one.<sup>21-23</sup> Entrance of the suppurative process into the canal is usually made by direct extension with necrosis of the bony wall. This applies particularly to infections of long standing where ample opportunity is afforded for resorption of bone to take

place. Extension through dehiscences in the bony canal and by way of tiny blood vessels<sup>8</sup> which perforate the canal has also been observed here.

A study of our histologic sections revealed that in a few cases the infection in the petrosa had destroyed the canal wall by osteoclastic resorption and had permitted the entrance of free purulent exudate into the lumen of the canal with the formation of a localized abscess (Fig. 3); in others it had continued on to produce a thrombosis of the carotid plexus of veins or to involve the carotid artery itself. Although extensive thrombo-arteritis of the carotid artery with necrosis of all the layers was observed in three cases, in not one of these did rupture of the vessel wall with extravasation of blood actually take place and in only one case was there metastasis to the brain. Reports have, however, appeared of patients who have died from hemorrhage of the carotid artery secondary to disease in the adjacent structures<sup>11, 13, 24, 25</sup> or secondary to trauma at operation (radical mastoidectomy,<sup>26</sup> petrous pyramid operation<sup>13, 27, 29</sup>). There was also reported a case of thrombosis of the carotid artery with secondary metastasis to the brain which followed chronic middle ear suppuration.<sup>18</sup>

New bone formation within the carotid canal is not an infrequent histologic finding in cases of petrositis (Fig. 4). It is usually associated with long standing infections in which extensive necrosis of the bony wall has taken place, and is observed most often on the inner aspect of the canal wall in close proximity to the lesion in the petrosa. Small blood vessels taking their origin in the inflammatory lesion in the petrosa can often be seen penetrating the bony canal and communicating directly with the focus of the new bone. Occasionally the new bone is encountered within the canal though the bony wall itself is apparently entirely intact and free of disease and the lesion in the petrosa is situated at a considerable distance from the canal. As the newly formed bone continues to grow inward it has been observed to gradually encroach upon the adjacent structures within the unyielding canal wall and on rare occasions to infiltrate the artery itself. Thus the carotid artery may become firmly united to the bony canal (Fig. 5). From the practical standpoint it is obvious that under these circumstances extreme difficulty would be encountered in separating the two when doing operations on the petrosa which entail this procedure (Ramadier's approach to the apex<sup>30</sup>). This opinion was previously expressed by Guild<sup>31</sup> and by Brovelli.<sup>4</sup> It must be remembered too that the carotid artery, not unlike other arteries in the body is, as a result of various constitutional factors, also subject to hyaline degeneration with calcific deposits in the vessel wall.<sup>32, 33</sup>

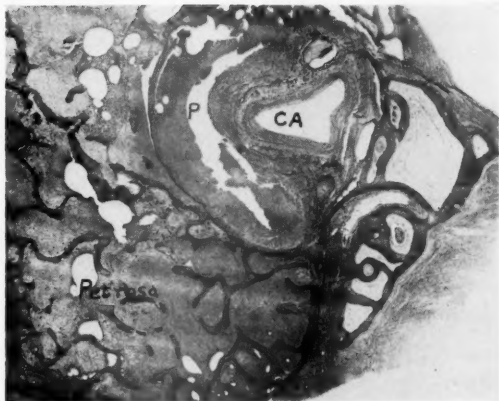


Fig. 3.—Photomicrograph showing necrosis of the bony carotid canal with purulent infiltration (*P*) in the lumen compressing the carotid artery secondary to petrositis. *CA* represents carotid artery.

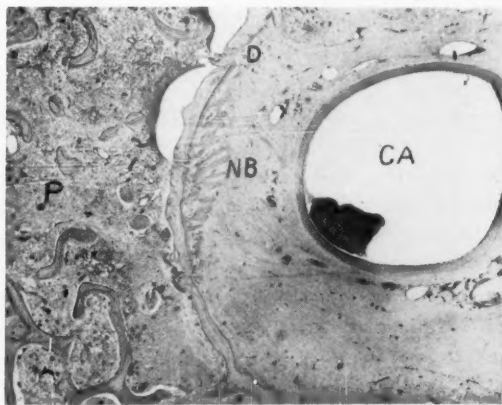


Fig. 4.—Photomicrograph showing new bone formation within the carotid canal secondary to suppuration of the petrous pyramid. Note multiple dehiscences (*D*) in the bony canal. *CA* represents carotid artery and *P*, petrosa.

*Relationship with the Jugular Bulb.* Since the carotid plexus of veins empties into the jugular vein, it is natural for inflammatory conditions, such as thrombophlebitis, occurring in the one to be readily transmitted to the other. The absence of valves in the venous plexus and the to-and-fro oscillatory movements of the blood<sup>34</sup> are probably factors in facilitating this spread of infection. It should be emphasized, however, that the thrombophlebitis may remain confined solely to the carotid plexus of veins and still give rise to a generalized blood stream invasion.

An infection in the jugular bulb can also enter the carotid canal by eroding the thin plate of bone which separates the two. Figure 6 demonstrates extension by this route in a case of thrombophlebitis of the jugular bulb.

*Relationship with the Cavernous Sinus.* The pericarotid veins form a continuous plexus with the cavernous sinus; it is understandable, therefore, why a thrombophlebitis of the cavernous sinus, irrespective of its cause or origin, is almost always associated with secondary inflammatory changes in the pericarotid veins. These changes have been seen histologically to vary in degree from that of a mild cellular infiltration of the perivascular tissues to one of extensive thrombosis of the vessels with diffuse purulent extravasation into the carotid canal and with secondary necrosis of the carotid artery. An example of pronounced inflammatory reaction within the carotid canal consequent to cavernous sinus thrombosis is portrayed in Fig. 7.

At the apex of the petrous pyramid the carotid canal is usually incomplete and does not entirely enclose the structures within. Thus the carotid artery and its plexus of veins lie more or less exposed to the adjacent meninges and are therefore readily subjected to the inflammations which usually occur in the meninges. Thrombophlebitis of the carotid plexus secondary to purulent meningitis, for example, is not an uncommon histologic finding.<sup>35</sup>

*Diagnostic Considerations.* The significance of keeping in mind the clinical diagnosis of thrombophlebitis of the carotid plexus of veins when dealing with otitic infections cannot be overemphasized. It has been pointed out by Tobeck,<sup>35,36</sup> Brunner,<sup>1</sup> and Druss,<sup>37</sup> that this condition may prove to be the sole etiological factor responsible for otitic sepsis complicating middle ear infection after all other causes have been ruled out. Similarly, as a complication of sinus thrombosis, it may prove to be the only cause for a continued sepsis which can not be accounted for on any other basis.

Of diagnostic value is the observation made by Eagleton,<sup>38</sup> namely, that thrombophlebitis of the carotid plexus of veins upon



Fig. 5.—Photomicrograph showing new bone formation within the wall of the carotid artery. Note that artery is intimately bound down to the adjacent bony canal. CA represents carotid artery; NB, new bone.



Fig. 6.—Photomicrograph showing section through the cochlea showing necrosis of the bony plate (*B*) separating the jugular vein from the internal carotid artery secondary to thrombosis of the jugular bulb. *C* represents cochlea; *CA* carotid artery; and *JB*, jugular bulb.



Fig. 7.—Photomicrograph showing suppurative process within the carotid canal with necrosis of the wall of the carotid artery and thrombosis secondary to cavernous sinus thrombosis. GG represents gasserian ganglion.

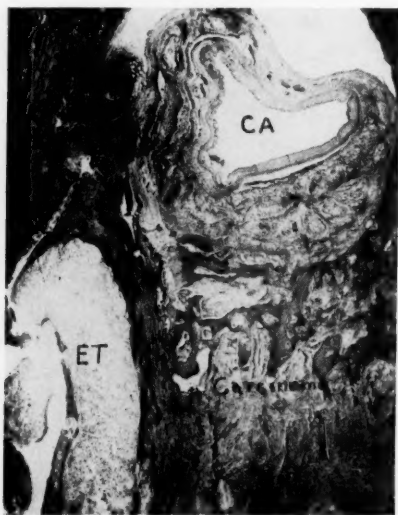


Fig. 8.—Photomicrograph showing carotid canal wall extensively destroyed by carcinoma. CA represents carotid artery and ET, eustachian tube.

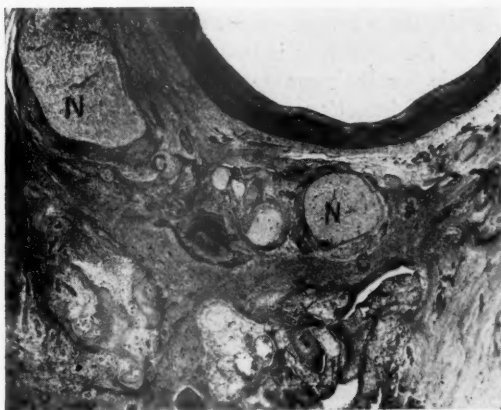


Fig. 9.—Photomicrograph showing carcinomatous infiltration of the perineural lymph spaces of carotid plexus of nerves. *N* represents nerve; *C*, carcinoma within lymph spaces.

reaching the cavernous sinus is usually associated with early and transient exophthalmos, but exophthalmos does not occur when extension to the cavernous sinus takes place by way of the petrosal veins. This is attributed to the fact that the carotid plexus joins with the anterior portion of the cavernous sinus at its junction with the ophthalmic vein, whereas the petrosals enter the posterior portion of the cavernous sinus and are not in close relationship with the ophthalmic vein.

*Neoplastic Extension.* Since neoplasms of the temporal bone and adjacent structures are also known to invade the carotid canal it was thought advisable to review the histologic findings of our cases with a view of possibly correlating this type of invasion with that of infections in general. There were three cases which showed such invasion, two by squamous cell carcinoma, and one by an embryonal cell carcinoma (Schminke tumor). In each of the three cases the tumor was found to have extensively destroyed the carotid canal wall and to have infiltrated the carotid plexus of veins (Fig. 8). The carotid artery itself was infiltrated by the tumor in two of the cases but the vessel wall was intact in each. Although rupture of the carotid artery with fatal hemorrhage secondary to neoplastic extension is a relatively rare occurrence, it has been recorded (de Reynier<sup>13</sup> and others).

Noteworthy in one of the cases of carcinoma studied, was the finding of a diffuse infiltration by tumor cells of the perineural lymph

spaces which surround the individual nerves in the carotid plexus (Fig. 9). While these lymph spaces could not be traced along the entire course of the nerves, it is strongly suggestive that they serve both as a vehicle for the propagation of tumor cells as well as for the transmission of infectious agents to other parts of the body.

#### SUMMARY

The carotid canal is subjected to invasion by suppurations within the adjacent structures. Some of the factors which play a role in this invasion and in the extension of the infection within the canal are considered. The histologic features of the carotid canal and its contents, and of the neighboring structures relevant to the spread of infections are presented. Emphasis is placed upon the significance of keeping in mind, in the treatment of otitic infections, the possibility of carotid canal involvement, particularly that of thrombophlebitis of the carotid plexus of veins.

1040 PARK AVENUE.

#### REFERENCES

1. Brunner, H.: Beitrage zur Kenntnis der otogenen Cavernosusphlebitis, *Monatssch. f. Ohrenheilk.* 60:2, 1926.
2. Lillie, H. I.: Summation and Conclusions of Symposium on Suppuration of Petrous Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44: 1129, 1935.
3. Kecht, B.: Die Bedeutung der Arteria Carotis interna in der Hals Nasen Ohrenheilkunde, *Arch. f. Ohr. Nas. u. Kehlk.* 143:1, 1937.
4. Brovelli, A.: Topography and Morphology of Internal Carotid Artery of Intrapetrosal and Intracranial Tract with Special Reference to Clinical Surgical Application, *Oto-rhino-laryng. ital.* 10:381 (Sept.) 1940.
5. Wolff, D.: Bilateral Atrophy of the Internal Carotid Artery. A Rare Anomaly, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 53:625, 1944.
6. Guild, S. R.: Normal and Pathological Anatomy of the Petrous Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:1011, 1935.
7. Wilson, J. G., Gaardsmoe, J. P., and Anson, B. J.: The Air Cells of the Petrous Portion of the Temporal Bone, *J. Laryng. and Otol.* 52:746, 1937.
8. Wilson, J. G.: Normal and Pathological Anatomy of the Petrous Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:1048, 1935.
9. Ferron, cited by Frank, I., and Sheer, C.: Sheath of the Internal Carotid Artery. Route for Infection from Primary Lesion, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 46:912, 1937.
10. Chorobski, I. J., and Penfield, W., cited by Eggston, A. A., and Wolff, D.: *Histopathology of the Ear, Nose and Throat*, Baltimore, The Williams and Wilkins Company, 1947, p. 107.
11. Politzer, A. A.: *A Text Book of Diseases of the Ear*, Transl. by Ballin, M. J., and Heller, C. L., Ed. 5, London, Balliere, Tindall & Cox, 1909.
12. Proctor, B., Lindsay, J. R., and Gonne, W. S.: Sequestration of Osseous Labyrinth, *Arch. Otolaryng.* 37:819, 1943.
13. de Reynier, J. P.: Les arrosions de la carotide interna dans son parcours intracranien, *Practa oto-laryng.* 8:562, 1946.

14. Proctor, B., and Lindsay, J. R.: Tuberculosis of the Ear, *Arch. Otolaryng.* 35:221, 1942.
15. Körner, O.: Das Fortschreiten von Erkrankungen der Paukenhöhle durch den karotischen Kanal, *Zeit. f. O.* 23:230, 1892.
16. Hessler, Jourdin, cited by Schlender, E.: Der Karotische Kanal als Überleitungsweg, *Monatssch. f. Ohr. Nas. u. Kehlk.* 63:666, 1929.
17. Lillie, H. I., and Williams, H. L.: Chronic Suppurative Lesions of the Petrous Pyramid (Report of Six Cases with Different Pathologic Characteristics), *Arch. Otolaryng.* 29:345, 1939.
18. Schlender, E.: Der Karotische Kanal als Überleitungsweg, *Monatssch. f. Ohr. Nas. u. Kehlk.* 63:654, 1929.
19. Jones, M. F.: Suppuration of the Petrosal Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:1036, 1935.
20. Fowler, E. P., Jr.: Normal and Pathological Anatomy of the Petrous Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:1056, 1935.
21. Lindsay, J. R.: Osteomyelitis of Petrous Pyramid of Temporal Bone, *Ann. Surg.* 122:1060, 1945.
22. Mullin, W. V.: The Fundamentals of Therapy. Symposium on Suppuration of Petrosal Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:1097, 1935.
23. Friesner, I., and Druss, J. G.: The Fundamentals of Therapy. Symposium on Suppurations of Petrosal Pyramid, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 44:1100, 1935.
24. Ballenger, W. L.: Diseases of the Nose, Throat and Ear. Revised by H. C. Ballenger, Ed. 5, Philadelphia and New York, Lea & Febiger, 1925, p. 323.
25. Barth, H.: Über eine chronisch verlaufende Pyramidenspitzenentzündung mit Blutung aus der A. carotis interna, *Zeit. f. Hals. Nas. u. Ohr.* 43:419, 1938.
26. Kopetzky, S. J.: Otolgic Surgery, New York, Paul B. Hoeber, 1925, p. 190.
27. Podestá, R., and Tato, J. M.: Histopathologischer Bericht über einen Fall von Petrositis (Operation nach Ramadier), *Acta Oto-laryng.* 25:254, 1937.
28. Richards, J.: The Petrous Pyramid. Its Surgical Anatomy and the Technique of the Operation for Its Removal, *Am. J. Surg.* 2:11 (Jan.) 1927.
29. Meyer, O.: Ein Beitrag zur Frage der Operation der Pyramidenspitzeiterungen, *Arch. f. Ohr.* 142:150, 1936.
30. Ramadier, J.: Exploration de la Pointe du Rocher Par Voie du Canal Carotidien, *Ann. d. Otolaryng.* 1:422 (Apr.) 1933.
31. Guild, S. R.: A Hitherto Unrecognized Danger in the Operation of Remadier for Suppuration of the Petrous Pyramid, *Acta Oto-laryng.* 25:561, 1937.
32. Dörfler, J.: Ein Beitrag zur Frage der Lokalisation der Arteriosklerose der Gehirngefäße mit besonderer Berücksichtigung der Arteria carotis interna, *Arch. f. Psychiatr.* 103:180, 1935.
33. dei Poli, G., and Zucha, J.: Beiträge zur Kenntnis der Anomalien und der Erkrankungen der Arteria Carotis interna, *Zentralbl. f. Neurochir.* 5:209, 1940.
34. Turner and Reynolds, cited by Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, The Williams and Wilkins Company, 1947, p. 187.
35. Tobeck, A.: Über die Einteilung der sog. Pyramidenspitzeiterungen, *Monatssch. f. Ohrenheilk.* 141:334, 1936.

36. Tobeck, A.: Schlagartge otogene Sepsis durch Thrombose im Plexus venosus caroticus im Anschlus an akute Pyramidenzelleiterung, Arch. f. Ohr. Nas. Kehl. 141:177, 1936.

37. Druss, J. G.: Thrombophlebitis of the Cavernous Sinus of Otitic Origin, Laryngoscope 52:115, 1942.

38. Eagleton, W. P.: The Fundamentals of Therapy. Symposium on Suppuration of Petrosal Pyramid, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 44:1113, 1935.

39. Eagleton, W. P.: Cavernous Sinus Thrombophlebitis, New York, Macmillan Company, 1926, p. 150.

## BLOOD PRESSURE CHANGES IN FENESTRATION

M. J. TAMARI, M.D.

AND

M. H. CUTLER, M.D.

CHICAGO, ILL.

Clinical studies using labyrinthine stimulation and experimental work on animals have shown that regular reflexes and reactions were produced causing definite changes of the vegetative system, such as increase of peristalsis, decrease of the basal metabolic rate,<sup>1</sup> and changes of the pupillary reactions.

In experimental studies on the influence of the vestibular apparatus on blood pressure, it has been shown by Spiegel and Demetriades<sup>2</sup> that caloric and rotatory stimulations of the labyrinth produce a lowering of blood pressure. The fall of the blood pressure started early with the stimulation and subsided slowly. In their studies on 55 rabbits fistulizations of the semicircular canals were omitted. Their deductions regarding this reflex phenomenon were registered by a special method marking the changes of blood pressure during and after rotation. This reflectory depression in blood pressure could also be produced after elimination of the vagus and sympathetic nerves. After cocainization of the middle ear, which produced a nonfunctioning labyrinth, the reflex phenomenon of depression of the blood pressure was completely absent.

Although the knowledge of these findings inspired many authors<sup>3</sup> to repeat and confirm the experiments, it still remained difficult because of objections to apply these reflexes to human vestibular functions.

The progress in labyrinthic surgery and especially the improved technique of fenestrations of the semicircular canals made it possible to repeat the experiment on a human with a nonpathologic labyrinth, to check and register the findings and to compare them with animal experimentations.

We have studied the blood pressure changes on 68 patients with a diagnosis of otosclerosis, who had been selected for fenestration. There were no cardiovascular diseases in this group and the emotional

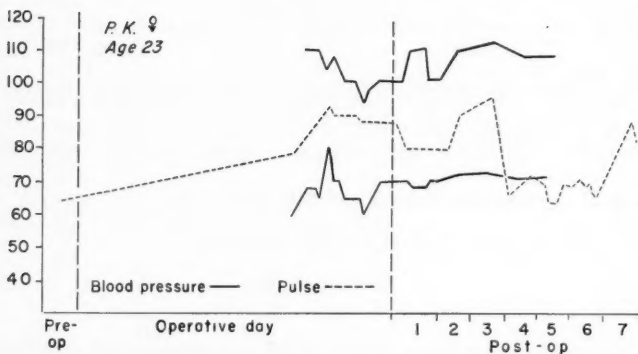


Fig. 1.—Lowering of blood pressure after fenestration and return to pre-operative high after three days.

types, as in some cases of tuberculosis, were not considered apt for these studies.

#### METHOD

The blood pressure was taken after the patient was admitted and observed for two days prior to surgery. Another reading was taken before the patient was prepared for surgery with drugs, and one just before surgery and continuously during surgery. It was signaled by the surgeon to the checking assistant at different times during the operative procedure and special attention was given to the opening of the canal at the ampullary end.

To avoid abnormal heat to the labyrinthine wall and to eliminate errors in stimulation which could be produced by using electrically driven burs or syringing the cavity with cold or hot water, we used gauges and chisels for the opening procedure of the ampulla. Both systolic and diastolic pressures were checked.

In a few cases the opening of the labyrinth was unknown to the checking assistant, and the surgeons were alarmed by the sudden change of the blood pressure.

As we stated before, we had an opportunity to study 68 patients with otosclerosis and two with Ménière's syndrome, who were undergoing labyrinthine surgery (fenestration). At the beginning of our observations the blood pressure changes were checked before and immediately after surgery for every half hour during the first 24 hours. In a large group of cases we found that the blood pressure was lowered one to three hours after surgery and remained on

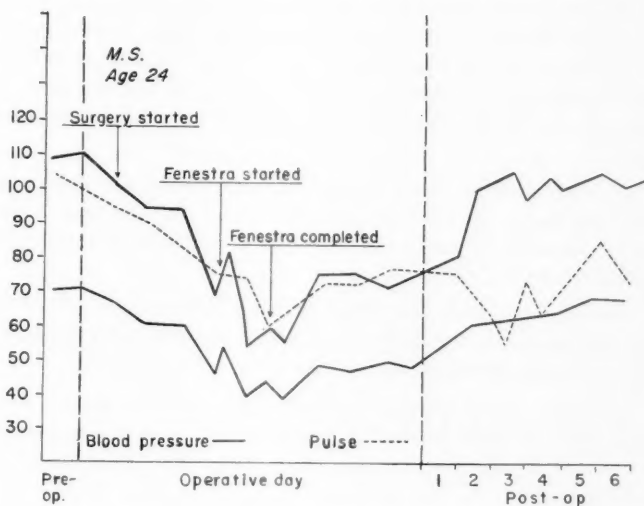


Fig. 2.—Blood pressure changes in a younger person during fenestration. Return to pre-operative normal after 24 hours.

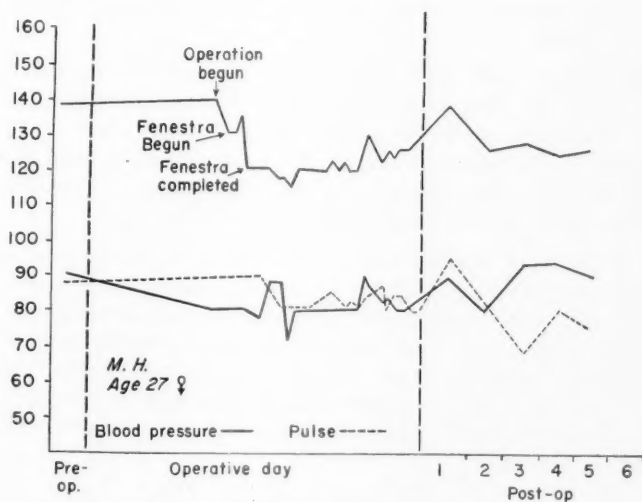


Fig. 3.—Lowering of blood pressure after fenestration. The depression lasted for three days in spite of vertigo and vomiting.

this lower level for 24 hours to 36 hours. It returned to pre-operative high on the third day after the operation, and the depression of the blood pressure persisted low mostly when other vegetative symptoms such as vomiting, vertigo, etc., were at the peak for the next three days and finally it returned to normal one week after surgery (Fig. 1).

Another type of blood pressure change was observed in a group of persons under 25 years of age. Here the characteristic drop occurred immediately after surgery was completed and the patient returned to bed.

The depression of the blood pressure lasted about 18-24 hours and returned to the pre-operative normal the day after surgery was performed and remained there (Fig. 2).

Another group showed a depression of 12-15 points in both systolic and diastolic phases for three or four days after surgery. It remained relatively low in spite of nystagmus, vertigo and vomiting (Fig. 3).

In the group operated upon in the last months we tried to check the blood pressure in all stages of surgery, namely, at the beginning of the incision, at the time the cortex was removed, at the opening of the antrum and removal of the bridge and finally during the procedure at the ampullary end of the horizontal semicircular canal.

In a few cases the blood pressure dropped during the manipulation of the fenestra and here, in two cases, the depression became alarming because the diastolic phase reached a low of 50. It returned to the pre-operative 80 and dropped in a typical manner to 54 one hour after surgery, and then returned to the normal pre-operative state of 120/80 after three hours (Fig. 4).

A group of patients with a relatively low pre-operative blood pressure of 100/70 showed comparatively moderate depression (10-12 points) after surgery. The blood pressure returned to the pre-operative level earlier than in the other groups, usually in 24 hours. In persons with a relatively high pre-operative blood pressure (170/110 - 185/120), the systolic drop after surgery reached 30-40 points below normal and returned to the pre-operative level the following day (Fig. 5).

#### DISCUSSION

Although it is well known that changes in blood pressure in man can be easily produced by psychic reactions, we would like to state that these studies were made on patients under local anesthesia

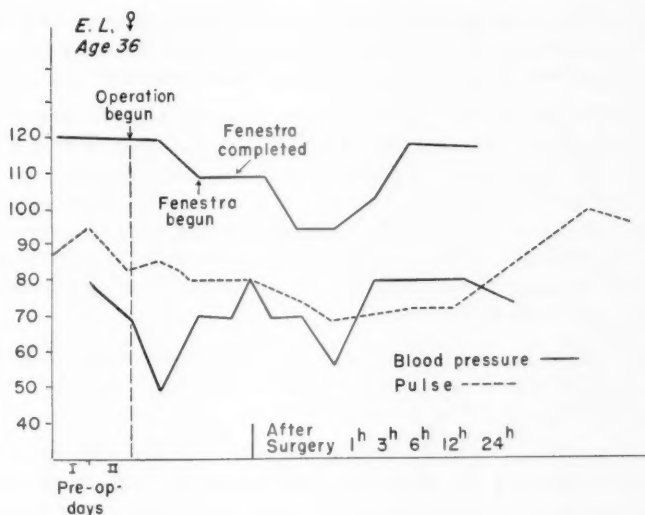


Fig. 4.—A deep depression of blood pressure during manipulation at the labyrinthine fenestra. Diastolic pressure below 50.

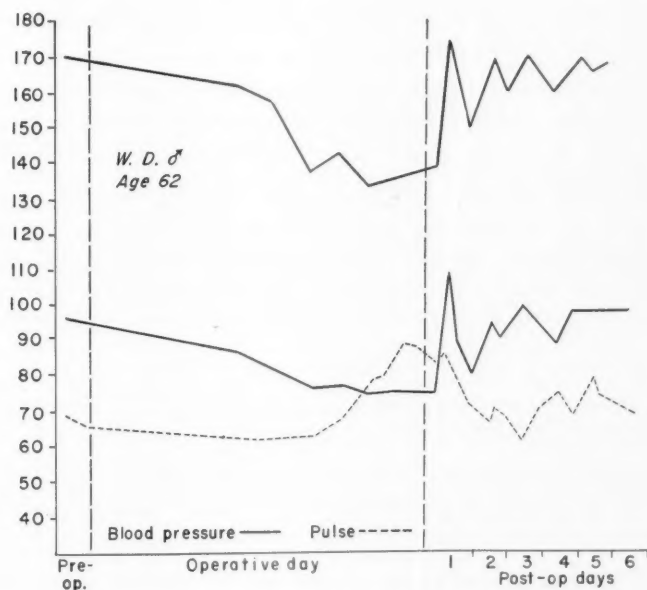


Fig. 5.—High pre-operative blood pressure. The normal high returned 24 hours after labyrinthine surgery.

or in twilight sleep and that pain, anxiety and excitement were controlled or eliminated. It is a well known clinical fact that emotions always produce a rise and not a fall in blood pressure.

The blood pressure does not seem dependent upon vertigo because it persisted after nausea and dizziness disappeared or else the patient still complained of dizziness and the blood pressure had already returned to normal. It was also experimentally shown by Spiegel and Demetriades that the blood pressure reflexes are present when the midbrain, the center of perceptive consciousness (*Bewusst-Werden von Empfindungen*), as well as the center of perception of vertigo was sectioned.

Objection to solely caloric clinical experiments arose with some writers because cold or hot water itself without labyrinthian stimulation may change blood pressure.

Also, rotations alone, which stimulated both labyrinths are mostly associated with vegetative reactions such as pallor, sweating, and palpitation. These are often connected with rising blood pressure.

There were also some characteristic changes in the pulse frequency and quality associated with the fenestration procedure.

Electrocardiographic studies are in progress and the results will be discussed at some future time.

Unchanged blood pressure after rotation and caloric stimulation in individuals with nonfunctioning vestibular apparatus was assumed by many authors but never verified clinically. In a group of youngsters between 10 and 16 years of age who became completely deaf and lost the labyrinthine function after meningitis, we were able to check the response of the blood pressure and found that there were no changes in blood pressure after whirling or syringing the ears with hot or cold water. The chart on the following page shows the blood pressure readings after rotation in these cases.

#### CONCLUSIONS

Blood pressure depressions have been produced by traumatic irritation of the labyrinth as in practiced fenestrations.

Blood pressure checks on persons with complete loss of labyrinthine function as in postmeningeal or luetic patients did not reveal any changes after caloric or rotatory stimulations.

The lowering of blood pressure occurs in characteristic types which depend upon the pre-operative blood pressure level. The depression is most manifest one hour after surgery has been completed.

BLOOD PRESSURE OF TOTALLY DEAF CHILDREN AFTER  
MENINGITIS; EFFECT OF ROTATION (10X, RT. OR L.)

NAME	BEFORE	AFTER	THREE MINUTES AFTER
Carolyn G.	120/78	124/78	122/78
Donna R.	124/92	126/92	126/88
Joanne B.	90/68	88/70	90/68
Joyce S.	108/84	112/84	108/82
Helen Y.	100/68	100/70	98/70
Lloyd E.	108/80	108/78	106/78
Paul H.	130/84	132/88	130/88
Ronald R.	112/78	110/82	112/78
R. S.	118/90	118/88	118/88
Sylvia R.	112/82	110/78	110/80

It generally persists for from one to three days independent of other labyrinthic reflexes and reactions.

25 EAST WASHINGTON STREET.

## REFERENCES

1. Exner, R., and Tamari, M.: Spirometrische Befunde Bei Labyrinth Reizung, *Ztschr. f. Neurologie und Psychiatrie* 132:235-263, 1931.
2. Demetriades, Th. D., and Spiegel, E. A.: Uber den Einfluss des Vestibular Apparates Auf den Blutdruck, *Ztschr. f. Hals-, Nasen- u. Ohrenh.* 3:220-225, 1922.
3. Talpis, L., and Wolfkowitzsch, M.: Zur Frage des Vom Labyrinth Ausgehendem Reflexes Auf Die Blutgefasse, *Monatsch. f. Ohren. u. Laryngo-Rhinologie* 62:1278-89, 1928.

## XVI

### SCLEROMA

#### RÉSUMÉ OF THE LITERATURE

#### REPORT OF THREE CASES

G. W. OLSON, M.D.

FRESNO, CALIF.

Scleroma is a chronic inflammatory disease of the respiratory tract of low contagiousness and long tenure. It is characterized by atrophic, hyperplastic and sclerotic changes in the mucous membranes of the nose, pharynx and larynx.

Recognition of the disease is attributed to von Hebra<sup>12</sup> who in 1870 described the first case. It remained for Mikulicz<sup>17</sup> in 1876 to recognize the typical foam cells and for von Frisch<sup>10</sup> in 1882 to find the supposed causative organism, Frisch's bacillus.

The disease is endemic in Southern Russia, Poland and Austria and is sporadic in Africa, Asia, Australia and the Americas.<sup>9</sup> It is believed that scleroma is not geographically limited, and that the incidence is steadily increasing in the Western Hemisphere.<sup>5</sup>

A compilation of published reports in our literature by Cunning and Guerry<sup>5</sup> in 1942 revealed 102 cases of scleroma occurring in the United States and Canada. Sixteen of these were in native born. Subsequently Dixon,<sup>8</sup> Dill,<sup>7</sup> Kellert,<sup>13</sup> Hara et al<sup>11</sup> and New et al<sup>10</sup> have reported 11 cases, of which 7 were in natives of this country.

Sivak<sup>21</sup> reported that scleroma is more common in females and that multiple cases are found often in families. Block<sup>2</sup> reported 128 cases seen in rural communities of the U.S.S.R. and he cited a familial incidence of 17.2%. The age of onset is usually in the third or fourth decade, indicating the low contagiousness of this disease.

The gross appearance of the lesions has been described as an atrophy, a hyperplasia, a sclerosis or a combination of these forms. The lesion may involve any part of the upper respiratory tract. Thus one can see that scleroma is a disease of protean nature. In the atrophic form there is an atrophy of the mucosa, hypersecretion and crusting. Early lesions in the hyperplastic form of scleroma are characterized by small, firm nodules usually found in the anterior nares. These nodules slowly increase in size, coalesce and gradually

spread posteriorly and down the respiratory tract, involving the pharynx, larynx and trachea. From the initial lesion on the mucosa the infection may progress to involve muscle, cartilage or bone with consequent disablement or disfigurement of the part involved. Chamberlin<sup>4</sup> observed that the three stages usually noted were the catarrhal, the proliferative and the cicatricial. Morrison<sup>17</sup> cited that from the clinical standpoint the three divisions were not so clear cut and that there is an overlapping of the stages.

The microscopic pathology has been well described by Mendiola.<sup>15</sup> In the early lesions there is an interstitial edema with many inflammatory cells and macrophages containing Frisch's bacillus, young fibroblasts and few new blood vessels. As the disease progresses there is a metaplasia of the mucosa with a change to a stratified squamous epithelium in nodular formation with hyperkeratinization of the germinal layers. From these germinal layers irregular prolongations of epithelium penetrate into the submucosa, anastomose and form connective tissue islands. Later most of the inflammatory cells disappear except for the macrophages, which clump together. A morphological and chemical change occurs by the action of Frisch's bacillus and these clumped macrophages increase in size and take on a round, oval or polygonal shape. Their cytoplasm is clear and the nuclei are round, with fine chromatophilic reticula, giving it the foam or lace-cell appearance. These are the Mikulicz cells, reported by Mikulicz<sup>16</sup> in 1877. Later a hyalinization of the cytoplasm occurs and hyaline bodies are formed. The few vessels found in this tissue have thick hyaline walls; and in old lesions the macrophages have been changed to fibroblasts and the collagenous material becomes hyalinized. As hyalinization occurs masses of this substance are encountered, 20 to 40 microns in size, which are called Russell or Unna bodies.

Frisch's bacillus (*Klebsiella rhinoscleromatis*) is generally considered to be the cause of scleroma, even though Koch's postulates have not been fully satisfied. Typical local lesions can be produced by injecting the organism into animals, but the disease cannot be transmitted in animals or man. Frisch's bacillus is a motile rod, often encapsulated, 1.5 by 3 microns in size, having rounded ends, which usually is found singly or in pairs. Frequently there is bipolar staining and the organism takes a gram-positive stain. This organism ferments the sugars, producing acid but no gas, and milk is not coagulated.

Sivak believes that scleroma is allied with atrophic rhinitis, and that certain exogenous influences will cause atrophic or hypertrophic tendencies. He noted in studies of families having scleroma that one

parent was involved in 75% of the cases, both parents in 33% and that 50% of the children showed an atrophic rhinitis, had positive serum reactions or were carriers of Frisch's bacillus. When these children were subjected to adverse conditions such as poor diet, crowding and unclean homes, Sivak thought that the bacillus could act specially and cause a secondary atrophic seropositive form of scleroma or develop the typical hyperplastic type. He believed that the two forms of the disease were pathologically opposed; that a low grade infection was followed by an atrophic form of the disease, and that a more virulent infection was followed by the hyperplastic form of scleroma.

The symptoms of scleroma vary with the part involved and the virulence of the disease. In the early stages there may be no symptoms. Usually the nose is the site of the oldest lesion and early symptoms are hypersecretion, obstruction and crusting. Later there may be epistaxis, fetid breath and complete obstruction with distension of the external nose. As the disease descends the respiratory tract, cough, soreness, hoarseness and dyspnea are noticed. Sivak<sup>20</sup> found that 80% of the 44 patients he treated had laryngeal involvement. Beck<sup>1</sup> reported a case having an esophageal lesion, which is uncommon.

The differential diagnosis should include atrophic rhinitis, syphilis, leprosy, mycoides, carcinoma and sarcoma. Frisch's bacillus can be found easily in the lesion and it grows readily on nutrient media. Biopsy of the lesion will establish the diagnosis if Mikulicz cells and the hyaline bodies of Unna are found, even though Frisch's bacillus cannot be isolated. Neuber<sup>18</sup> noted that the specificity of Frisch's bacillus could be determined and that it could be differentiated from other encapsulated bacilli by complement fixation and agglutination tests. An allergic reaction can be obtained in the patient with scleroma by the use of specific antigens. Testing is done by intracutaneous injection which is followed by a reddened edematous halo in 24 hours, succeeded by a hard, circumscribed infiltration lasting three or four weeks.

The treatment of scleroma has been rather unsatisfactory. Mercury, arsenic, iodine, arsphenamine, tuberculin, sulfonamides, fever therapy, excision and galvanic cautery are a few of the medicaments and procedures which have been used with some success. Neuber<sup>18</sup> stated that the treatment of scleroma offered no difficulties when an autovaccine or specific convalescent serum was used. Unfortunately, few others have obtained his results. He used the smallest amount of the autovaccine which would give an allergic reaction by intracutaneous injection as the initial dose. Slightly larger amounts were

given every four or five days for 12 or 15 doses. The course was repeated two or three times after six or eight weeks had elapsed. Sivak reported fair success from irradiation and less success with autogenous vaccines and attenuated cultures. Denegri<sup>6</sup> reported a series of 39 cases treated by irradiation with 50% good results in cases of high localization and 3% good results when the lesions were low in the respiratory tract. Weiss<sup>22</sup> observed that the histologic changes in scleroma following intensive irradiation were (a) extensive fibrosis, (b) increased hyalinization, (c) decreased cellular content of the stroma and (d) disintegration and disappearance of the Mikulicz cells. Denegri reported that the rays were not always bactericidal for in some of his patients treated by irradiation with good results the organisms could still be demonstrated in the nasal secretion. He felt that the tissue changes produced by irradiation rendered conditions for the growth of the bacillus less favorable. Most observers feel that irradiation is the treatment of choice for, as Canfield<sup>3</sup> stated, it hastens the end result of scleroma, which is sclerosis, with less disability of the parts involved. In 1947 Hara et al<sup>11</sup> first reported the use of streptomycin in the treatment of scleroma. Two of their five patients failed to improve following irradiation and subsequent use of streptomycin caused marked regression of the disease. They used 0.5 gm in aerosol inhalations and 2 gm intramuscularly in divided doses each day. New et al<sup>10</sup> reported another case treated with streptomycin. This patient had been diagnosed in Shanghai, China, in 1938 and had been treated with autogenous vaccines with improvement until 1945, at which time the case was reported as cured. When she was seen late in 1946 at the Mayo Clinic there was marked bilateral nasal obstruction, scarring of the pharynx and granulomatous masses on the epiglottis and left larynx with considerable glottic obstruction. The organism cultured was found to be inhibited by 0.3 unit per cc of streptomycin. The patient was given 250 mg of streptomycin intramuscularly every three hours. Side reactions to the drug forced its discontinuance two times. The total dosage given was 97.25 gm in 50 days and this effected a clinical cure.

The prognosis varies with the stage of the disease and virulence of the organism. A few cases have been known to have spontaneous arrest;<sup>14</sup> others have had long remissions and one case was cured following typhus. In the literature scleroma is usually described as a slowly progressing granuloma descending the respiratory tract, ending in death from an intercurrent infection.

#### REPORT OF CASES

CASE 1.—B. N., a housewife, aged 33, born in Mexico, was first examined on April 26, 1943, having been referred for tonsillectomy.

The patient had spent her first 11 years in seven towns in Mexico, moving to this country in 1920. She has resided in ten cities in three western states. The family history revealed that her mother had been subject to violent fits of sneezing and profuse nasal discharge called "catarro crónico" for many years. One sister died at the age of 19 from pulmonary tuberculosis. Her past history was negative, and her only complaints were sore throats following infrequent attacks of coryza. Examination revealed the tonsils to be enlarged and the anterior pillars were injected. The pharynx appeared normal. Anterior rhinoscopy disclosed an irregular, hyperplastic mass 2 x 1.5 cm in size attached to the medial surface of the right inferior turbinate near the anterior end of the middle turbinal. The mass contacted the septum and was covered by a thin crust. The tumescence had the appearance and the consistency of the hyperplasias often noted on the middle turbinate in allergic rhinitis. The remainder of the nasal mucosa was slightly hypersensitive in appearance. No pus was found on shrinkage and transillumination of the anterior paranasal sinuses was clear. The ears, nasopharynx and larynx were normal.

The tumor mass was removed by snare and biting forceps. Little bleeding was encountered. The patient did not return for re-examination until August 29, 1944. She denied having any further symptoms. Rhinoscopy revealed a nodular growth the size of a pencil eraser at the site of the original lesion. The nasal mucosa was still somewhat hypersensitive and the remainder of the examination was normal.

The recurrent mass was removed with snare and biting forceps. The original prepared section, undiagnosed the year previously, and the recurrent tissue were reported as follows:

"October 18, 1943. This is a chronic infective granuloma. The numerous Russell-Plimmer bodies are not as conspicuous as they would have been in a preparation with a stronger eosin stain. There are also occasional large pale cells with centrally placed shrunken nuclei. These features suggest rhinoscleroma.

"September 2, 1944. This inflammatory lesion is a chronic infective granuloma having all of the attributes necessary for a diagnosis of rhinoscleroma. The granulation tissue is covered by attenuated, stratified, squamous-celled mucosa. In the granulation tissue there are numerous fuchsinophil (Russell-Plummer) bodies, both nucleated and non-nucleated. There are also many large cells with pale, reticulated cytoplasm and irregular pyknotic nuclei. These are the Mikulicz cells, characteristic of the granulation tissue of rhinoscleroma prior to the stage of dense cicatricial sclerosis."

The patient was referred for irradiation therapy. She was given treatments at weekly intervals totalling 550 roentgen units, with a target distance of 10 in., 1-mm aluminum filter, 85 K.V.; 5 ma; 22 roentgen units per minute for five minutes a treatment. Re-examination every three months since has shown the nose to be normal.

CASE 2.—L. G., aged 30, a single woman of Mexican nativity (sister of B. N. in Case 1) employed in the housekeeping department of a hospital, was examined on September 7, 1944, along with all members of B. N.'s immediate family.

She admitted no symptoms referable to the upper respiratory tract, although later she said that occasionally there was a clear nasal discharge.

Examination of the throat revealed nothing abnormal. Anterior rhinoscopy disclosed a polypoid mass 1 x 1 cm in size in exactly the same location as was her sister's. The tumor was pale, smooth and had a rubber-like consistency on palpation. The nasal mucosa was normal, no pus was noted on shrinkage and transillumination showed normal findings. The ears, nasopharynx and larynx were likewise normal.

The tissue was removed completely by scalpel. Microscopic examination was reported as follows:

"September 12, 1944. Rhinoscleroma in a relatively early stage, with the characteristic granulation tissue present underneath a stratified squamous mucosa. Fibrosis and cicatrization have not yet developed." (Fig. 1.)

The patient was referred for irradiation therapy and she received the same dosage as did her sister. She was not seen again until August 12, 1947, at which time there were no symptoms but a small nodule had recurred at the original site. This was removed with the scalpel. The microscopic examination was:

"August 25, 1947. This specimen shows the characteristic granulation tissue of rhinoscleroma. In this area the process is still in the vascular prefibrotic stage."

A second course of irradiation of the same dosage was given and no recurrences have been noted on quarterly examinations.

CASE 3.—G. B., a housewife aged 29, born in New York of Greek descent, was seen on January 7, 1944. The chief complaints were severe nasal obstruction and crusting. The family history revealed that her family and that of her husband were descendants

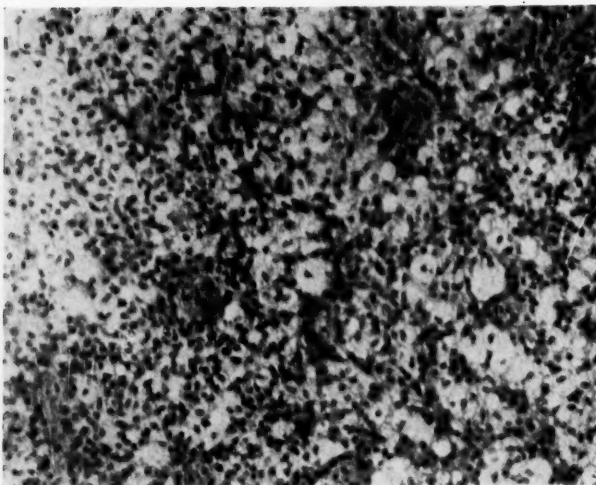


Fig. 1.—Photomicrograph showing characteristic granulation tissue of rhinoscleroma in the early, prefibrotic stage. (Hemalum and eosin stain.)

of the Athenian colonizations of the Black Sea coast. For the first 20 years of her life she either lived with relatives who were natives of Turkey or she had many Turkish relatives visiting with her. She knew of none who had chronic nose or throat complaints.

The present illness began in July, 1930, when she developed a severe right ear ache which lasted seven days, unassociated with otorrhea. On the fourth day of the otalgia the patient blew her nose at which time a "huge bloody pulp" was expelled from the right nostril. No further symptoms were noted until January, 1931, when she had another right ear ache. At this time she first noted a nasal discharge, and soon afterward a tendency to crust formation and obstruction. During the summer of 1931 she had slight attacks of epistaxis daily. The nasal crusting and obstruction gradually became so pronounced that in 1932 she was forced to give up a singing career. She visited many physicians during the next ten years but she did not carry out any of the various treatments and operations advised. The only relief during this time was obtained with saline nasal irrigation. In May, 1943, a submucous resection of the nasal septum was performed and for the next four months there was no nasal obstruction, although the crusting continued and its removal by irrigation caused considerable soreness. The right nostril was said to have been completely closed by September, 1943.

This patient was first seen by me on January 11, 1944. A round nodular mass could be seen filling the right vestibule of the nose. It was attached by a broad base to the nasal septum and was firm, immobile and nontender. Rhinoscopy through the left nares revealed a small anterior septal perforation 4 cm from the columella. The anterior margin of the perforation was indurated and palpation revealed it to be included in the tumor. No septal cartilage could be palpated near the perforation. The left nasal mucosa appeared somewhat atrophic. No pus was found on shrinkage and transillumination was clear. The ears, nasopharynx, pharynx and larynx were normal. A tentative diagnosis of chondroma was made and surgical removal was advised. The patient did not return until September 22, 1944, at which time she decided to have the tumor removed. The mass had now extended through the anterior nares and distended the right ala.

Extirpation of the tumor was done under local anesthesia. A mucosal incision paralleling the mass anteriorly and superiorly allowed the tumor to be peeled away from the columellar cartilage anteriorly. Farther back the septal cartilage was found to be involved, therefore it was removed along with most of the mucosa lining the septal perforation. Hemorrhage was slight, and the tumor was found not to involve the floor of the vestibule. The nasal mucosa on the right side was more atrophic than that on the left. A second and smaller mass was noted on the lateral wall of the nose in front of the anterior end of the inferior turbinate. This lesion was also widely dissected. Microscopic findings were:

"October 16, 1944. The specimens from the three locations specified all show a chronic infective granuloma with the characteristics of rhinoscleroma. The mass from the septum shows the oldest process and in this there is less of the characteristic foam-celled granulation tissue, and more fibrous connective tissue." (Fig. 2.)

The postoperative course was uneventful and epithelization occurred normally. She was referred for irradiation therapy and she received the same course as did the patients in Cases 1 and 2, consisting of 550 roentgen units in five weekly treatments.

Six months following surgery she moved to Southern California, and there developed hay fever. After the onset of this allergy she had no more nasal crusting. The hay fever was severe enough to warrant desensitization treatment. On re-examination every three or four months, the nose was found to be free of scleroma although the membranes were hypersensitive.

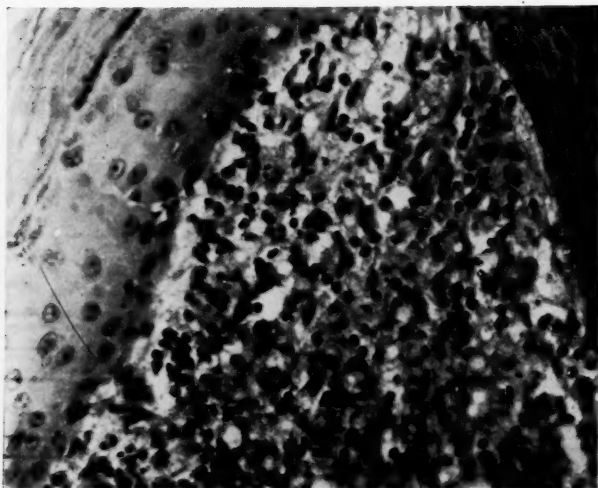


Fig. 2.—Photomicrograph of section showing more fibrous connective tissue and less of the characteristic foam-celled granulation tissue.

#### SUMMARY

A review of the literature revealed that 112 cases of scleroma have been reported in the United States, and of these 23 were native born. This report adds three more cases, one of which (Case 3) was born in and has never left this country.

Scleroma is believed to be a disease afflicting those people who live in unhygienic and crowded surroundings. The three cases reported here occurred in middle-class people and the factors of poor diet, uncleanness and crowding were not applicable to them.

When hyperplastic nasal mucosal lesions are found in a person who has contacts with people from areas where scleroma is prevalent, a painstaking differential diagnosis should be made. This can be done by biopsy, culture, complement fixation and agglutination tests. Two of the cases herein reported (1 and 2) show scleroma in an early stage with no symptoms and few clinical findings.

Case 3 was unusual in that the advent of hay fever apparently changed the atrophic mucosa into a typical allergic rhinitis. It is unlikely that the irradiation was responsible for the sudden cessation of the nasal crusting nine months following therapy. It is also unlikely that the tissue changes in atrophic rhinitis would be reversible

to the hyperemic and hypersecretory phase. One might speculate that nasal allergy may act as a deterrent to the progression of scleroma. Those cases characterized by a prolonged and benign clinical course may have been so modified by the co-existence of a subclinical nasal allergy.

In early and relatively benign scleroma such as observed in these three reported cases, good results have been attained by surgical excision followed by irradiation therapy.

Streptomycin may become the treatment of choice if subsequent usage affords as spectacular results as in the few cases which have been reported.

The tissue examination in the three cases herein reported were made by Carl V. Weller, Professor of Pathology, University of Michigan. He did not prepare the original tissue in Case 1, but did report the findings from the prepared slide.

1759 FULTON STREET.

#### REFERENCES

1. Beck, J., in discussion of paper by Weiss, J. A.: Rhinoscleroma: Histologic Changes after Telerradiation; Review of Scleroma in the United States, *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 48:531-534 (June) 1939.
2. Block, S. G.: Familial Scleroma of the Upper Respiratory Passages, *Zhur. Ush., nos. i gorl. bolez.* 15:157-158, 1938.
3. Canfield, N.: Case of Rhinoscleroma in a Native Born, *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 42:903-908 (Sept.) 1933.
4. Chamberlin, W. B.: Rhinoscleroma. Is it an Indigenous Disease? *Arch. Otolaryng.* 23:285-294 (Mar.) 1936.
5. Cuning, D. S., and Guerry, Du Pont, III: Scleroma, *Arch. Otolaryng.* 36:662-668 (Nov.) 1942.
6. Denegri, J.: Relates oficiales de II Congress Sud Americans de Otorrhinolaryngolazia 1:221-228 (Nov. 8-12) 1944.
7. Dill, P. J.: Rhinoscleroma; Case, *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 52:496-500 (June) 1943.
8. Dixon, F. W.: Scleroma, *Arch. Otolaryng.* 36:937-939 (Dec.) 1942.
9. Figi, F. A., and Thompson, L.: Rhinoscleroma, *J. A. M. A.* 91:637 (Sept. 1) 1928.
10. von Frisch, A.: Zur aetiologie des Rhinoscleroma, *Wien. med. Wchnschr.* 32:969-972, 1882.
11. Hara, H. J., Pratt, O. B., Levine, M. G., and Hoyt, R. E.: Scleroma: A Clinico-Pathological Study of Seven Cases in One Family, *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 56:769-783 (Sept.) 1947.
12. von Hebra, F.: Ueber ein eigenthümliches Neugebilde Naserhinosklerom, *Wien. med. Wchnschr.* 20:1-5, 1870.
13. Kellert, E.: Rhinoscleroma, *New England J. Med.* 229:647-650 (Oct. 21) 1943.
14. Knapp, E.: Incidence of Scleroma in Germany; Report of Four Cases, *Ztschr. f. Hals-, Nasen- u. Ohrenh.* 45:67-76 (Mar. 14) 1938.
15. Mendiola, R.: Histopathology of Scleroma of the Upper Respiratory Tract, *Laryngoscope* 56:677-686 (Nov.) 1946.
16. Mikulicz, J.: Weber das Rhinoskleran, *Arch. f. klin. Chir.* 20:485, 1876.

17. Morrison, L. F.: Scleroma (Rhinoscleroma). Report of Three Cases, *Arch. Otolaryng.* 28:531-537 (Oct.) 1938.
18. Neuber, E.: Serologic and Allergic Reaction of Scleroma and Its Specific Management, *Monatschr. f. Ohrenh.* 2:58 (Feb.) 1940.
19. New, G. B., Weed, L. A., Nickols, D. R., and Devine, K. D.: Rhinoscleroma Apparently Cured with Streptomycin, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:412-417 (June) 1948.
20. Sivak, M.: Scleroma in Slovakia, *Monatschr. f. Ohrenh.* 75:55 (Feb.) 1941.
21. Sivak, M.: Scleroma in Slovakia, *Monatschr. f. Ohrenh.* 75:183 (April) 1941.
22. Weiss, J. A.: Scleroma (Rhinoscleroma): Histologic Changes Following Teleradium Therapy, *Arch. Otolaryng.* 30:38-49 (July) 1939.

## XVII

### THE TREATMENT OF BELL'S PALSY WITH HISTAMINE

D. A. SKINNER, M.D.

NEWARK, OHIO

Bell's palsy, commonly called rheumatic facial paralysis or refrigeration palsy, is a peripheral paralysis of the facial nerve due to vasospasm<sup>4</sup> of the stylomastoid artery, its branches, and anastomoses. The vasospasm is primary; however, the occurrence of edema of the nerve may cause further compression, thus complicating the vasospastic picture. Kettel<sup>6</sup> has suggested the actual cause of the paralysis as being one of ischemia; the compression in the facial canal is only a secondary phenomenon and not the real cause of the paralysis. In cases of peripheral paralysis other than those of Bell's palsy, vasospasm is secondary.

Among other causes of peripheral paralysis of this nerve are constitutional, exanthematous and virus diseases; focal infections; hemorrhage; tumors; surgical procedures in the region of the facial nerve; and trauma. Peripheral paralysis resulting from any of these conditions is not Bell's palsy. However, Bell's palsy is the most common type of peripheral paralysis.

Interchangeable use of the terms Bell's palsy and facial paralysis, due to other causes, are found in many earlier articles and books. Sir Charles Bell<sup>2</sup> first described the classic symptoms of peripheral facial paralysis in 1826. At that time, the term Bell's palsy was used for all peripheral facial paralyses. Bell's palsy has often been cited as being of idiopathic origin. Partial or complete recovery occurs within a period of a few weeks to several months, up to 15 months, or else the paralysis is permanent.

The onset of the paralysis is usually sudden. The close proximity of the facial nerve and the stylomastoid artery to the canal wall probably accounts for the suddenness of paralysis in some individuals after exposure to cold. In many cases, the patient wakes up in the morning with the paralysis, which is often first discovered by looking in the mirror. This condition usually occurs in an otherwise healthy individual but in some cases a slight cold or an acute infection precedes the onset of the paralysis. Vestibular testing with cold water, the use of the ice collar, dental extractions,

and sudden fright have been thought to be precipitating factors in rare cases. Titus<sup>15</sup> has personally observed the onset of two cases of Bell's palsy. He says, "It was just as though they had been shot. Both patients had been sitting in a draft in extremely cold weather and the face suddenly twisted to one side."

The exciting factor appears to be exposure to cold in about 70% of the cases. Some follow severe emotional upsets but, in many cases, the exact precipitating factor remains unknown. Nervous, emotional, and/or vascular disturbances are present in a large percentage of cases. If the patient has a vasospastic tendency, it would appear that the excitant, in most cases, is due to exposure to cold, while in others it may be similar to the pathogenesis of vascular headaches. Undoubtedly, physical, psychosomatic, inhalant, and food allergies play a role. The lack of balance of the endocrine glands may be the basis for a great many of these vascular disorders.

The facial deformity is due to the immobility of the muscles supplied by the facial nerve. This is characterized by the inability to raise the eyebrow, to wrinkle the skin of the forehead, to close the eye completely, and to move the lip and cheek. On attempting to close the eyes, the lids of the affected side remain open, and the eyeball of that side moves upward and outward. This is described as Bell's phenomenon. Epiphora develops, due to the loss of power to move the lids, a slight ectropion, and paralysis of Horner's muscle. The face appears masklike and expressionless. The nasolabial fold is obliterated. The corner of the mouth has a tendency to sag and is slightly drawn over to the opposite side. Whistling is impossible and speech imperfect because the lips cannot be properly closed.

Bell's palsy is usually unilateral. Nicol<sup>7</sup> has reported one bilateral case. Textbooks consider this to be rare. Most cases of facial diplegia that have been described are not Bell's palsy.

At the onset, pain may or may not be present. Involvement is usually worse where the pain is severe and of long duration and, if present, is usually within the ear, in the temple, mastoid regions and at the angle of the jaw. It is usually of a vasodilating character, but in a few cases it may be similar to the pain of a geniculate neuralgia. The duration of pain, if present, is usually three or four days and seldom over ten days, but may last for a longer period.

It must be remembered that a small part of the facial nerve is sensory. As the block approaches the geniculate ganglion, pain may be present affecting the sensory component of this nerve, giving pain in the eye, temple, cheek, and in the mastoid region. This is a chronic

deep-seated pain, which may last for several hours or days without subsiding. Geniculate ganglion neuralgia<sup>11</sup> often follows Bell's palsy.

Females<sup>3</sup> are affected more frequently than males because, as a rule, they are subject to more vascular disturbances. No age group is exempt. If we can divide the population into three age groups of 25 years each, it is the second group, the 25 to 50-year group, that is more frequently affected.

Recurrences of this condition are not uncommon. Familial incidences may occur, especially where vascular disturbances are present. The onset occurs more commonly in the spring and fall seasons.

More left facial nerves are affected than right. The right ear, as a rule, is more protected than the left while sleeping or driving a car. Rather large external auditory meatuses have been observed with little or no cerumen in cases where refrigeration was a factor. A small, curved external meatus with hairs and cerumen aids in protection against cold. The chilling of the contents of the facial canal probably occurs through the external auditory meatus, creating a vasospastic condition. Other nerves<sup>13</sup> of the body are not so affected by exposure to cold, due to the fact that they have no close proximity to the exterior, and are better protected by soft tissues.

Diplacusis occurs when there is involvement of the stapedial nerve. An increased hearing sensitivity and discomfort to certain loud noises are experienced following paralysis of the stapedial muscle. Perversion of taste takes place on involvement of the chorda tympani. One, both, or neither of these nerve branches may be affected. Diplacusis and loss of the sensation of taste, if present, usually persists for only a few days. This early restoration<sup>5</sup> is probably due to a more abundant collateral blood supply which is present near the knee of the nerve.

The site<sup>16</sup> of the lesion of the peripheral part of the facial nerve can be localized at different levels by the presence or absence of taste and tears.

If the block is below the exit of the chorda tympani, there is no disturbance of taste or tears. Above this exit and below the geniculate ganglion there is a loss of taste with no effect on the tears. If the lesion is in the region of the geniculate ganglion, there is both loss of taste as well as partial or complete loss of tears. Proximal to the geniculate ganglion, taste is not affected and there is a partial or complete loss of tears only.

## SUMMARY BY TSCHIASSNY

LOCATION OF LESION	PRESENCE OF	
	TASTE	TEARS
Infrachordal	Yes	Yes
Suprachordal	No	Yes
Transgeniculate	No	No
Suprageniculate	Yes	No

Over half of our cases, according to the above classification, have been of the suprachordal type.

The authors of many textbooks and articles have considered Bell's palsy to be due to neuritis, secondary to a focus of infection, while others have thought that it might be due to a virus. Foci of infection may or may not be present and, if so, the treatment of the same does not seem to alter the paralysis. Ballance and Duel<sup>1</sup> have considered it to be a neuritis with edema and they have advocated early decompression and slitting the nerve sheath.

Kettel<sup>6</sup> has considered this condition to be a vascular disturbance of the *vasa nervorum*, where the blood supply of the nerve has been blocked. On the basis of 50 cases, he found that in 56% the nerves were unquestionably edematous, in 4% possibly atrophic, and in 2% the continuity of the nerve had been interrupted. In 20% of his cases, bony necrosis of the mastoid process was present, and in 36% there was some necrosis of the wall of the facial canal. In half of these cases, the bony necrosis had spread to the entire cellular system and, in the other half, it was present only in the apex of the mastoid process, especially around the stylomastoid foramen. In some of the cases, a clear dark fluid was observed in the mastoid cells but no pus or granulation tissue was present. These observations<sup>14</sup> have not been confirmed by others in this country.

Histamine diphosphate is one of the best vasodilators. It increases the blood supply and helps to smooth out the vessels. The treatment<sup>12</sup> was started in 19 cases of Bell's palsy by giving subcutaneous injections of histamine diphosphate every two or three days.

In 17 cases, treatment was started by using low-dose therapy, usually starting with 0.1 cc of a 1:10,000,000 dilution and gradually increasing until the optimum effective dosage was reached.

## ANALYSIS OF 19 CASES

---

DATES OF ONSET	
January	1
March	3
April	5
May	1
June	1
August	2
September	3
October	3
SEX	
Males	5
Females	14
AGE	
Minimum age	19
Maximum age	67
AGE GROUP	
Under 25 years	3
25-50 years	13
Over 50 years	3
ETIOLOGICAL FACTORS	
Exposure to cold	14
Emotional upsets preceding	7
Vascular phenomena	10
Acute infection	4
PREVIOUS ATTACKS	2
EAR INVOLVED	
Right ear	4
Left ear	15
SIZE OF AUDITORY CANAL	
Large	13
Medium	6
Presence of cerumen	3
PRESENCE OF PAIN	15
DURATION OF PAIN WHEN PRESENT 2-40 days	

When improvement occurred, we aimed to keep the patient on that amount with very little change of dosage.

In two cases, treatment was started by using high-dose therapy, starting with 0.1 cc of a 1:10,000 dilution and gradually increasing to 1.0 cc of a 1:10,000 dilution. In these two cases, improvement did not occur before a period of one month. We thought a more rapid and phenomenal response had been effected by using the low-dose type of therapy.

Intravenous histamine (2.75 mg dissolved in 250 cc normal saline) was administered in one case where the low-dose method only gave a partial response. No further improvement was noted.

Nicotinic acid is probably one of the most commonly used vasodilators. When this drug is transformed into an amide, vasodilatation is brought about. Nicotinamide cannot be substituted for this use since it does not produce the necessary vasodilatation. Rosenberger<sup>10</sup> has used nicotinic acid for peripheral paralysis of the facial nerve of herpetic origin. In one case where there was little response to low-dose histamine therapy, we administered nicotinic acid before meals, just enough to produce a "flush," for a period of three weeks before returning to low-dose histamine.

Dihydroergotamine tartrate is a sympathetic paralyzing agent that is commonly used for the treatment of migraine, histaminic cephalgia, and obscure head and neck pains. This drug gave considerable relief from pain in one of our cases.

In addition to vasodilating therapy, the following were advocated:

1. Mineral oil eyedrops.
2. Hot compresses and massage of the face with the fingertips from below upward, for five minutes three times a day.
3. Strapping of the face with cellulose tape or facial splints.<sup>8, 9</sup>
4. Treatment of the foci of other infections.
5. Electric stimulation given once a week, in the cases where there is not an early response.

In 16 cases, or 84%, the rapidity of improvement was phenomenal. In this group, the shortest recovery occurred in four days, the longest in 48, and the average was 19 days.

The paralysis recurred in two patients when dosage was increased above the optimum effective amount. However, complete restoration took place when the proper effective dosage was again

repeated. In one case, paralysis did not recur when the dosage was increased far above the optimum effective amount. Following complete recovery in another case, 50 mg of pyribenzamine caused a slight recurrence of the paralysis.

The degree of improvement is stated as follows:

- 0=no improvement
- 1=25% improvement
- 2=50% improvement
- 3=75% improvement
- 4=complete improvement

#### IMPROVEMENT

	TO WRINKLE FOREHEAD	TO CLOSE EYE	TO LAUGH, SMILE, AND WHISTLE
Cases 1-16	4	4	4
Case 17	4	4	2
Case 18	4	4	2
Case 19	3	4	3

#### SUMMARY

Inasmuch as Bell's palsy is a vasospastic disease, therapy must be directed towards vasodilatation or "breaking the block."

The vasodilators and sympathetic paralyzing drugs may be interchangeably used in many cases with good results.

We have preferred the low-dose histamine therapy to the high-dose and intravenous methods.

The accomplishment of vasodilatation by drugs may eliminate some cases from surgical decompression and slitting of the nerve sheath.

The value of electric stimulation, in some cases, must not be overlooked.

The results obtained in the treatment of Bell's palsy with vasodilators have been encouraging. Although these observations have been made in only 19 patients, the results were so striking that the effect of this type of therapy appears unquestionable.

25 EAST LOCUST STREET.

## REFERENCES

1. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy, *Arch. Otolaryng.* 15:1 (Jan.) 1932.
2. Bell, Charles: On the Nerves of the Face, *M. Class.* 1:155 (Oct.) 1936.
3. Connole, Joseph V.: Vaccine Treatment of Facial Paralysis, *Pennsylvania M. J.* 44:467-469 (Jan.) 1941.
4. Hansel Foundation: Course in Ophthalmologic and Otolaryngologic Allergy.
5. Hilger, Jerome A.: The Nature of Bell's Palsy, *Laryngoscope* 59:228-235, 1949.
6. Kettel, Karsten: Bell's Palsy, *Arch. Otolaryng.* 46:437-472 (Oct.) 1947.
7. Nicol, A. A. McIntosh: A Case of Bilateral Bell's Palsy, *Brit. M. J.* 1:220 (Feb. 20) 1943.
8. Pickerill, H. P. and Pickerill, C. M.: Early Treatment of Bell's Palsy, *Brit. M. J.* 2:457, 1945.
9. Pracy, J. P.: Facial Splint for Treatment of Bell's Palsy, *Brit. M. J.* 1:528 (Apr. 6) 1946.
10. Rosenberger, Harry C.: Herpes Zoster Oticus with Facial Paralysis and Acoustic Symptoms, A Subjective Experience, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 50:271 (Mar.) 1941.
11. Ross, Otho B., Jr.: Geniculate Ganglion Pain, *North Carolina M. J.* 10:114-117 (Mar.) 1949.
12. Skinner, D. A.: The Use of Histamine in Bell's Palsy, *Ohio State M. J.* 45:565-566, 1949.
13. Skinner, D. A.: The Role of Vasospasm, *Digest Ophth. and Otolaryng.* 11:746-775 (Oct.) 1949.
14. Tickle, Thomas G.: The Repair of Facial Paralysis of Otic Origin, *Surg. Clin. North America* 136:438-445 (Apr.) 1948, New York number.
15. Titus, Norman E.: The Early Use of Electrotherapy in Bell's Palsy, *M. Rec.* 155:169, 1937.
16. Tschiasny, Kurt: The Site of the Facial Nerve Lesion in Cases of Ramsey Hunt's Syndrome, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 55:152 (Mar.) 1946.

## XVIII

### OTOSCLEROSIS IN IDENTICAL TWINS

A REVIEW AND REPORT OF TWO ADDITIONAL PAIRS

ARTHUR L. JUERS, M.D.

CHICAGO, ILL.

Inasmuch as environmental factors in some instances seem to influence the acceleration of deafness progression in cases of clinical otosclerosis, a number of cases of otosclerosis in identical twins have in recent years been studied and reported by several authors with the hope of providing some cause-effect relationship between progression, heredity and environment. This discussion will review the contributions to date and report an additional two sets of identical twins.

*Genetics as Related to Otosclerosis.* Guild<sup>1</sup> has estimated that histologic evidence of otosclerosis occurs in about 8% of the population. In 81 temporal bone sections which had such histologic changes, the otosclerotic lesion had produced ankylosis of the stapediovestibular articulation in only ten. Inasmuch as seven "silent" lesions are present to every one in which otosclerotic fixation is manifested clinically, Guild concludes that eugenic measures to control otosclerosis in future generations are rather futile. While otosclerosis has been classed as a dominant hereditary trait by some authorities, others<sup>2</sup> have considered it as being recessive. The difficulties encountered in determining whether it is a dominant or recessive trait become apparent when we consider the proven fact that there are approximately seven parents or offspring with a "silent" histologic lesion to every one with clinical manifestations of the disease. In a recent study of the incidence of a family history of deafness in cases of clinical otosclerosis, Smith<sup>3</sup> found that in a small series of women only about one-third gave a history of familial deafness. In this same group 37% gave a history of their deafness being initiated by, or, if present, increased by pregnancy.

*General Studies on Identical Twins.* Newman<sup>4</sup> made an extensive study of identical twins with particular attention to the influence

---

From the Department of Otolaryngology, Northwestern University Medical School, Chicago, Illinois.

Accepted as a candidate's thesis by the American Laryngological, Rhinological and Otolological Society, Inc.

of environment on twins living together or apart from early infancy. In general, the more obvious physical characteristics were influenced but little by environmental differences. On the other hand, evidence was presented that education has a definite influence on ability. However, in such a study it must be remembered that it is extremely difficult to assess environments which, though on an

TABLE 1.

Family History	Set 1		Set 2	
	6 brothers and sisters—4 of whom have deafness. Father, grandfather, one aunt and uncle deaf. None on maternal side.		One brother and sister — normal hearing. No familial deafness.	
	Twin A	Twin B	Twin A	Twin B
Birth Weight	7¼ lb.	7¼ lb.	5 lb.*	6½ lb.
Early Feeding and Diet	Early breast fed. Average diet.	Same	Same	Same
Colds	Infrequent	Infrequent	Average	Average
Measles	No	No	Age 3—no otitis	Same
Mumps	No	No	Age 5	Age 5
Pertussis	No	No	Age 2—Residual bronchitis 1 yr.	Age 2—Prompt recovery
Tonsillitis	Attack — Age 21 No tonsillectomy	Attacks age 15 Tonsillect. age 16	Occas. sore throats Onset age 15. Tonsillect. age 26	No tonsillectomy. Sore throat — 3 wk. at age 16.
Other Illness	None	R. inguinal abscess age 10. Incised.	Hysterectomy—age 42. Fibroid.	Cesarean section, age 38. Placenta previa.
Menses			Onset 12½ — Irreg. for 6 mo.	Onset 13 yr. Reg.
Pregnancy			Age 24. Heart attack. Mitral stenosis with emboli immediately after. In bed several months. Fever at first.	Four pregnancies. First at age 21. Last, age 38.
Deafness Onset Noted	Age 18	Age 13	Age 24 during early postpartum period.	No deafness observed to date.
Tinnitus	Both ears	Left ear	None	None

\*Weight equal at one year.

economic level seemingly different, may on the basis of fundamental cultural, nutritional and psychological criteria be very much alike. It is not a simple task to select those environmental factors which may be most significant in altering the maturation and general development of a given individual. Newman has classed as "nature" those influences on the genesis of the individual which are based on

the genetic factor and as "nurture" those which are dependent on the prenatal and postnatal environment. One of the earliest of the prenatal factors which may create inequality in identical twins is the matter of fetal circulation. It is well known that the relative size of twins is directly proportional to the size of their respective placentas. (Whether the growth and nutritional needs of the fetus influence the size of the placenta or whether the placental size determines the size of the fetus is not known.) Variations in birth weight of identical twins may differ by as much as one or two pounds. It is usually presumed that the smaller twin has grown less in size because of less intra-uterine nutrition. Whether this difference in size is determined by a genetic factor or is merely a chance of early environment is not known. Irrespective of the reason for the common difference in size, it would seem likely that the smaller twin would be at a slight disadvantage in overcoming adverse environmental situations in the early weeks of life and that this factor could, in some instances, very well contribute toward differences later in life. This may be of significance in the second set of twins reported in this paper.

Snyder<sup>5</sup> states that "environmental influences have a strong effect on many bodily processes, including even, in some cases, the morphological processes of development, as when an insect or mammal develops faulty skeletal structures through dearth of some necessary ingredient, or has faulty reactions of the central nervous system because of some injury to it or misuse of it, despite the fact that it may have had genes which under better circumstances would have led to a properly proportioned skeleton or to exemplary behavior. A given gene, however, is never to be thought of as literally being a gene for this or that character, but only as a gene with given possibilities of reaction, affording the potentialities for the development of certain characters provided external conditions as well as the assemblage of other genes present with it are suitable." In commenting on previous concepts of heredity he states that "the most objectionable of all were beliefs that if a certain trait is hereditary it cannot also be influenced by the environment and, conversely, that if it is influenced by the environment it cannot at the same time be hereditary." Newman states that "each character is an expression of an interplay of hereditary and environmental factors. Each character must be studied as a separate unit, for environmental differences may well modify some characters far more than others."

A very striking example of the influence of environment on development is the fetal anomalies which may result when the mother has German measles in the first trimester of pregnancy. Deafness,

cataract and cardiac anomalies are particularly prone to occur. The usual explanation given as to why the fetus is especially vulnerable during early pregnancy is that the basic structures or anlage are formed during this time. It has been postulated that the particular system affected will depend somewhat on which area is undergoing the fundamental differentiation at the time the infection occurs.

While this is an extremely obvious instance of the genetic influence being altered by environment, undoubtedly there are many similar situations in which the immediate effect is not apparent. According to Newman and Snyder such environmental modification of the basic genetic control occurs even after birth. They have presented convincing evidence that ability and personality are influenced by environment and education.

With these thoughts in mind it will then be of interest to make an analysis of identical twins with clinical otosclerosis to determine

TABLE 2.

	Set 1		Set 2		Normal Range mg/100 cc
	Twin A	Twin B	Twin A	Twin B	
Cholesterol	265.0	234.0	263.0	271.0	Men-150-240 Women-180-260
Calcium	12.1	10.9	12.7	11.8	9.5-11.0
Phosphorus	3.35	2.93	3.16	2.84	2.5-4
Phosphatase	3.85	4.53	4.04	3.97	1.5-4 units

whether factors other than heredity seemingly influence the genesis of otosclerosis.

*Previous Reports on Otosclerosis in Identical Twins.* There have been 15 sets of twins reported in which there was a diagnosis of clinical otosclerosis. The first such a set was reported by Albrecht.<sup>6</sup> Since then additional cases have been reported by Rodin,<sup>7</sup> Shambaugh,<sup>8</sup> Hagens,<sup>9</sup> Fowler<sup>10, 11</sup> and Rosenberger.<sup>12</sup> Albrecht and Rodin observed no essential difference in the onset or course of the deafness in either member of their respective pair. Shambaugh reported three sets of twins. In each set the one with greater cold susceptibility seemed either to develop deafness earlier or have a greater degree of deafness. In the pair reported by Hagens deafness was first noted in the twin who had the first pregnancy. Fowler has reported eight sets of twins but observed no consistent relationship between cold susceptibility and onset or severity of deafness. One

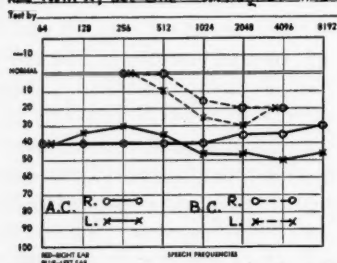
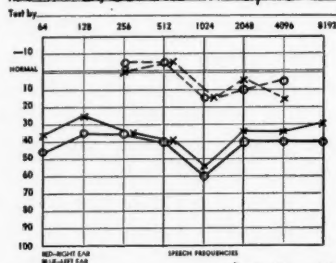
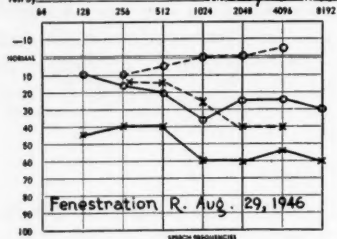
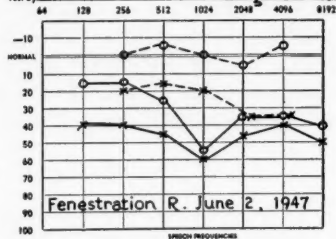
set reported by Fowler is unusual in that one twin has blue sclera whereas the other twin has white sclera. There is no history of fragile bones. The one with blue sclera has an early stapes fixation type of deafness and the other twin has normal hearing except for a 10-db loss for the lower three frequencies in one ear. This must be considered as possible very early evidence of stapes fixation in this ear. There was no apparent difference in environment for these twins. In his last report he stated that "the thing that surprised me most over the years is the tendency in the great majority of cases for the lesion to stabilize and this in spite of child bearing." In the set reported by Rosenberger the one with more colds seemed to have slightly poorer cochlear function as evidenced by the bone conduction level. Fowler did extensive blood chemistry studies on his cases and observed no significant deviation from normal. However, the phosphatase activity seemed in general to be slightly less in the twin who first noted deafness.

#### REPORT OF CASES

Two additional sets of identical twins with otosclerosis are being reported in this paper. In the first set the history and clinical course show only minor differences. In the second set one noted deafness at the age of 24, and the other at the age of 43 has observed no hearing impairment.

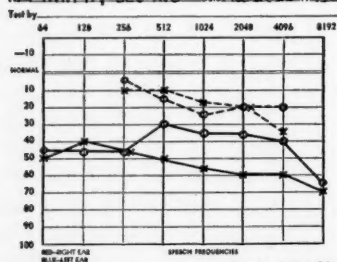
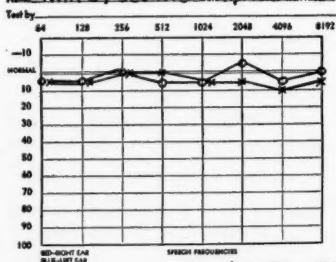
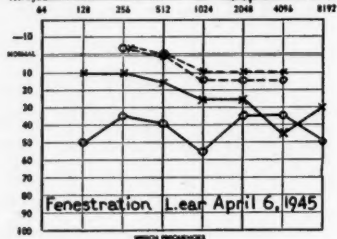
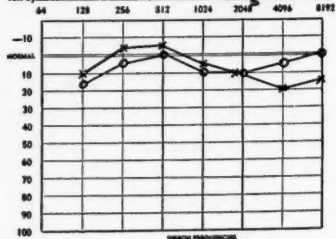
SET 1.—These 29-year-old male identical twins were first examined in 1946. As will be observed in Table 1, twin B noted the onset of his deafness five years before twin A. Twin B had an inguinal abscess incised at the age of 10 and was slightly more subject to tonsillitis beginning at the age of 15. Because of this and his deafness he had a tonsillectomy at the age of 16. Both have had a fenestration done. Incidentally it is of interest to note that two years after operation there is a slight upward shift in the bone conduction acuity in the operated ear of each twin (right ear) as compared with the slight downward shift in level on the unoperated ear (see Audiometric Charts 1 and 2). The possible significance of this will not be discussed here.

SET 2.—These 43-year-old female twins are unusual in that one developed clinical otosclerosis while the other has maintained essentially normal hearing. There is no question as to the identical nature of this pair because they have always been mistaken for each other, and have identical features such as hair, eye color, ear contour, etc. One has an unusually short big toe on the left foot and the other has the same variation on the right. This type of asymmetry is, according to authorities such as Newman,<sup>4</sup> particularly characteristic

Name Twin A, Set One Date Aug. 27, 1946Name Twin B, Set One Date May 26, 1947Name Twin B, Set One Date May 26, 1947Name Twin A, Set Two Date July 15, 1949Name Twin B, Set Two Date Aug. 18, 1949

Audiometric Chart #1

Audiometric Chart #2

Name Twin A, Set Two Date March 26, 1945Name Twin B, Set Two Date April 24, 1945Name Twin A, Set Two Date Sept. 20, 1949Name Twin B, Set Two Date Aug. 23, 1949

Audiometric Chart #3

Audiometric Chart #4

of single ovum twins. Their history before marriage revealed the following significant differences as is tabulated in Table 1:

1. Twin A (the one with deafness) weighed  $1\frac{1}{2}$  lb. less at birth.
2. Twin A had a residual bronchitis for a year after pertussis whereas twin B recovered promptly.

Twin A has had one pregnancy. Immediately after delivery she developed a severe heart attack which was diagnosed as a mitral valve lesion. Some emboli were noted. Deafness was first observed at this time. The deafness slowly progressed and was diagnosed as clinical otosclerosis. A fenestration was performed and a 22-db level of hearing has been maintained for four and one-half years. Twin B has had four pregnancies, but still has essentially normal hearing (see Audiometric Charts 3 and 4).

The blood chemistry studies on these two sets of twins revealed no significant differences between the twins or deviations from normal (Table 2). Since otosclerosis is a localized lesion, it is not surprising that no evidence of general metabolic derangement has been found by the usual chemical and clinical tests now available.

*General Aspects of Otosclerosis.* Circulatory disturbances in and around otosclerotic foci have long been observed by investigators in the study of otosclerosis. Wittmaack<sup>13</sup> in particular has studied this aspect in detail. Nager<sup>14</sup> describes otosclerosis as a "localized osteodystrophy which represents the reaction of the otic capsule to one or different hitherto unknown irritative causes." He believes that "it seems very likely that the 'blue mantles' observed histologically represent the very first alterations of the otosclerotic process of unknown etiology and distributed by the blood stream into the otic capsule. A small blood vessel is located in the center of each 'blue mantle'." As far as can be determined clinically, Nager has observed that heredity may not be as important as has been believed in the past. He also believes that pregnancy has been overestimated as a stimulating factor in the progression of deafness.

In discussing the various reports concerning the significance of the fissula ante fenestram with respect to the frequent occurrence of otosclerosis at this point, Bast and Anson<sup>15</sup> state that "these reports agree that the fissular area is one of histological instability, and that its defective, or delayed, ossification is an important factor in the formation of otosclerotic bone." Pathologic bony foci in other areas "were of the branching type, apparently following vascular channels."

Kosokabee<sup>16</sup> has found that unossified cartilage in the fissular area is present in 80% of women and 50% of men. This would coincide somewhat with the difference in incidence of clinical otosclerosis in women and men.

Lempert and Wolff<sup>17</sup> have recently reported further on histological studies of ossicles removed during fenestration from patients with clinical otosclerosis. On the basis of their observations they believe that the initial pathological changes leading eventually to otosclerosis are within the bloodstream. The pathological alterations which they observed in the walls of the vessels were believed to result in metabolic disturbances and structural changes in the bone around the pathological vessels. If further comparable control studies on ossicles from temporal bones without otosclerosis reveal no such vascular changes, then the evidence presented by Lempert and Wolff will have added significance. Should this theory of origin prove valid, then the apparent absence of general metabolic disturbance can be readily understood. The cause of the localized vascular pathology still remains to be determined. Whether the vascular changes produce the alterations in the bone or whether the local metabolic disturbance precedes changes in the vessel walls can be determined only by further detailed studies such as reported by Lempert and Wolff.

#### COMMENT

In view of the suggested relationship between local circulatory disturbances and the genesis of otosclerotic lesions, the apparent onset of deafness at the time of a major cardiovascular upset in twin A of Set 2 may be particularly significant. An illness such as this would undoubtedly have some effect on any area predisposed toward instability and/or in which the vascular bed was especially susceptible to deleterious environmental influences. In the event that a minor unnoticed degree of deafness was present before delivery, the rather sudden progression of deafness from that time on would suggest that this illness had at least a definite accelerating effect on the otosclerotic process. Twin B of this set will be watched with considerable interest for any future clinical evidence of otosclerosis.

The differences in the history of twin A and B of Set 2 would suggest that twin A had less ability to overcome adverse environmental situations. Whether this reflects a slightly different genetic potentiality or whether minor environmental differences early in life caused a constitutional inadequacy in twin A is a matter of speculation. In view of Newman's studies<sup>4</sup> on identical twins, a logical explanation of the differences in twin A and B would be that their

final physiological and physical pattern was the result of an interplay of genetic and environmental influences, the latter being primarily responsible for the differences. The prolonged bronchitis after pertussis and the heart lesion after delivery in twin A would in all probability be considered as environment. It is possible that environmental influences in twin A initiated activity in a latent otosclerotic process whereas in twin B there has not until the present time been sufficient acceleration of the process to manifest itself clinically. Whether a histological lesion exists in twin B or whether the very slight drop in a few frequencies audiometrically is evidence of beginning otosclerotic activity is, of course, not known.

The differences in the family history of Set 1 and Set 2 are striking. In Set 1 there is a history of strong hereditary predisposition on the paternal side. In Set 2 there is no family history of deafness. We might postulate that with a strong predisposition in Set 1 (having a larger, more unstable fissular area?) otosclerotic activity began with less apparent environmental stimulation; on the other hand, that in Set 2 (having a smaller, more stable fissular area?) a greater environmental stimulating factor was necessary to initiate activity and that in twin B of Set 2 insufficient impetus has been given to produce clinical manifestations of otosclerosis. These ideas must, for the present, remain in the realm of speculation.

#### CONCLUSIONS

1. The general aspects of environment and heredity as related to identical twins are briefly discussed.
2. Previous reports of otosclerosis in identical twins are reviewed.
3. A brief résumé of the present knowledge of the genesis of otosclerosis is made.
4. Two additional sets of identical twins with otosclerosis are reported. In one set, one member has no definite evidence of clinical otosclerosis.

55 EAST WASHINGTON STREET.

#### REFERENCES

1. Guild, S. R.: Histologic Otosclerosis, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 53:246-266 (June) 1944.
2. Gates, R. R.: *Human Heredity*, New York, Macmillan Company, 1946.
3. Smith, H. W.: Effect of Pregnancy on Otosclerosis, *Arch. Otolaryng.* 48: 159-170 (Aug.) 1948.
4. Newman, H. H., Freeman, F. N., and Holzinger, Karl J.: *Twins, Study of Heredity and Environment*, Chicago, University of Chicago Press, 1937.

5. Muller, H. J., Little, C. C., and Snyder, L. H.: *Genetics, Medicine and Man*, New York, Cornell University Press, 1947.
6. Albrecht, W.: Über Konstitutionsprobleme in der Pathogenese der Hals-, Nasen- und Ohrenkrankheiten. 2. Die Otosklerose, *Ztschr. f. Hals-, Nasen- und Ohrenh.* 29:55-60, 1932.
7. Rodin, F. H.: Identical Hearing Defect in Identical Twins, *Arch. Otolaryng.* 17:179 (Feb.) 1933.
8. Shambaugh, G. E., Jr.: Otosclerosis in Identical Twins, Report of Three Sets, *J. A. M. A.* 104:1216 (Apr. 6) 1935.
9. Hagens, E. W.: Otosclerosis in Identical Twins, *Arch. Otolaryng.* 34:583-587, 1941.
10. Fowler, E. P.: Otosclerosis in Identical Twins, Three Case Histories, *Laryngoscope* 52:718-731 (Sept.) 1942.
11. Fowler, E. P.: Otosclerosis in Identical Twins, Five Case Histories, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 56:368-378 (June) 1947.
12. Rosenberger, Harry C.: Otosclerosis in Identical Twins, *Arch. Otolaryng.* 38:594-596 (Dec.) 1943.
13. Wittmaack, K.: Die Ursache der Otosklerose: Ein Vorschlag zur ursachlichen Behandlung, *Arch. f. Ohren- Nasen- u. Kehlkopfh.* 129:150, 1931.
14. Nager, Felix R.: Pathology of the Labyrinthine Capsule, and Its Clinical Significance, *Nelson Loose-Leaf Medicine of the Ear*, E. P. Fowler, Jr., 1947, pp. 237-270.
15. Bast, T. H., and Anson, B. J.: *The Temporal Bone and the Ear*, Springfield, Chas. C. Thomas, 1949, pp. 263-290.
16. Kosokabee, H.: Ueber die Knorbelfugen in der Labyrinth Kapsel beim Embryo, Kinder und Erwachsenen und Über Deren Zusammenhang mit der Entstehung des Otosclerotischen (Oto-spongiotischen) Stuttgart, Herdes, 1922.
17. Lempert, Julius, and Wolff, Dorothy: Otosclerosis: Theory of Its Origin and Development, *Arch. Otolaryng.* 50:115-155 (Aug.) 1949.

## XIX

### THE NASAL TIP IN RHINOPLASTY

#### USE OF THE INVAGINATING TECHNIQUE TO PREVENT SECONDARY DROPPING

GUSTAVE B. FRED, M.D.

BOSTON, MASS.

Every surgeon who performs cosmetic rhinoplasty realizes that the success of the operation depends largely on his skill in modeling the tip of the nose and on his ability to ensure the permanence of the new configuration. Once the alar cartilages have been shaped correctly, the remodeled nose may, at the operation's end, look completely satisfactory. But all too often this early result fails to stand the test of time, and the tip eventually sags, taking on a rounded, pendulous shape that is no less disappointing to the surgeon than to the patient. This sagging of the tip is the end-result of downward traction exerted by the linear contracting scar at the suture line between the columella and the septum and of a downward pull of the orbicularis oris muscle of the lip attached to the columella. In addition to the sagging, there develops too high a profile line, because the cartilaginous dorsum of the septum has not been lowered enough at the time of the operation to compensate for the sagging tip.

To compensate for the tendency of the tip to "drop," most surgeons, during the operation, raise or overcorrect it to a higher level than they expect or intend it to have ultimately. This they do by using the orthopedic stitch of Joseph<sup>1</sup> high on the septum and low on the columella and lowering the cartilaginous dorsum of the septum to the level to which they wish the tip eventually to fall, after final healing has taken place.

If one could accurately judge, at the time of the operation, how much or how little postoperative change might occur in the tip's position, there would be no problem. But one cannot anticipate the exact degree of recession, since it varies so widely in different indi-

---

From the Departments of Surgery and Otolaryngology, Beth Israel Hospital, Boston, Mass.

Presented as a candidate's thesis to the American Laryngological, Rhinological

viduals. In some the change is clearly perceptible; in others no change at all occurs. The solution of the problem lies, of course, in the perfecting of some technique that would give reasonable assurance that the nasal tip would remain permanently in the position it has at the end of the operation.

For the past two years, I have been using a procedure which does exactly that, with highly satisfactory results. To this procedure I have given the name *invaginating technique*.

#### THE INVAGINATING TECHNIQUE

After the nasal hump is removed, in the course of the typical cosmetic rhinoplasty, the cartilaginous ventral surface of the septum is lowered. Next, one resects a wedge of the septum, its base on the nasal dorsum, so that the nose can be shortened the desired amount. Through the transfixion incision between septum and columella, the caudal edge of the septum over its entire anteroposterior extent is denuded of its mucous membrane one-eighth of an inch, on both sides, to septal perichondrium or bare septal cartilage. A periosteal elevator or small knife and scissors are used.

The cut edge of the columella is then everted to one side by means of a fine double hook, and with a No. 15 Bard-Parker blade one makes a linear vertical incision in the exact center of the columella at its lower or posterior half to a depth of slightly less than one-eighth of an inch. This shallow groove never extends to the medial crura cartilages and certainly should not extend into the area between them. One black silk suture between the septum and columella (straight septocolumellar suture)<sup>2</sup> invaginates the denuded cartilage of the caudal edge of the septum into the prepared pocket in the columella. The narrow skin flaps of the columella will be seen to encircle or clasp the denuded cartilage of the septum. The septal mucous membrane and the skin edges of the columella fit snugly together and all raw edges are approximated. Two mattress sutures of No. 000 catgut on a straight needle through the columellar skin flaps and septal cartilage still further maintain the parts in their new position. One may, in addition, if so desired, also unite the skin edges of the columella and mucous membrane edges of the septum with No. 0000 catgut on a small, curved atraumatic needle.

One of the gravest mistakes in rhinoplasty is to shorten the nose too much. The result is a retracted columella and an unnatural-looking nasal tip. Since the invaginating technique shortens the nose by another eighth of an inch, the surgeon who uses it must make certain that when he shortens the nose by the conventional method of removing a wedge of septal cartilage from its caudal edge he

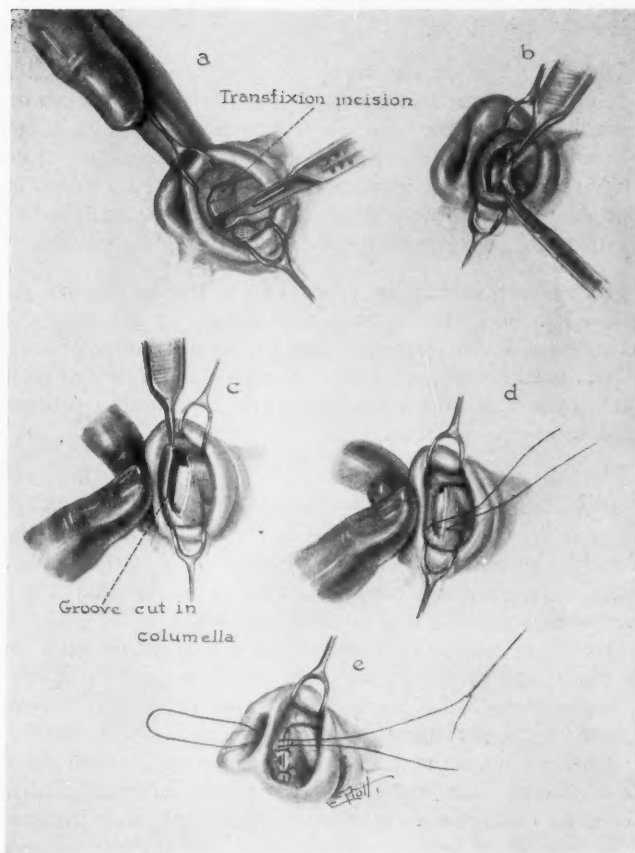


Fig. 1.—The Invaginating Technique.

- a. Columella separated from nasal septum through a transfixion incision.
- b. Removal of one-eighth inch of mucous membrane from caudal edge of left side of nasal septum. Right side is similarly treated through right nostril.
- c. Shallow groove made in exact center of cut edge of columella.
- d. Bare edge of septal cartilage invaginated into groove in columella. Columellar edges can be seen to encircle septal cartilage.
- e. Mattress sutures through both columellar skin edges and septal cartilage, fixing position of columella to the rigid septum and preventing secondary dropping of tip.

removes an eighth of an inch less of the septum than the length the nose is ultimately to be.

The advantage of the invaginating technique over the simple septocolumellar suture in maintaining the tip in its new position lies in the adherence of the columella's encircling flaps, over a comparatively wide area, to the perichondrium remaining on the septal cartilage or to the bare septal cartilage, depending on which is used, so that the healing traction is exerted in a cephalic or upward direction rather than downward toward the philtrum of the lip.

Some objection may be raised to the use of the invaginating technique on the ground that it might result in too rigid a lobule. Experience has shown, however, that the tip loses none of its mobility. The reason is that such a small portion—one-eighth of an inch—of the columella is drawn back onto the septum that enough of it remains to ensure mobility.

The literature contains a number of papers<sup>3, 4</sup> on the nasal septum describing a procedure by which the entire quadrangular septal cartilage is dissected clear of the nose, reshaped and then reinserted between the septal mucous membrane flaps. The freely mobile septal cartilage is then brought far forward into a bed between the medial crura of the columella and sutured into place. The effect of this procedure is to bring the septal cartilage too far forward between the medial crura cartilages, with the result that the tip is too rigid. The invaginating technique differs in that the mobile columella is only to a very slight degree drawn backward onto the immobile septum; furthermore, no septal cartilage is inserted between the medial crura cartilages. The septal cartilage and medial crura cartilages do not come in contact with each other. Since only such an extremely small section of the septum is invaginated into the soft tissues of the columella, enough columella remains to supply a mobile tip.

After the invaginating sutures have been tied, a lateral osteotomy is performed to narrow the nasal bones. Modeling of the tip is the last step. In the congenital cosmetic rhinoplasty, the septocolumellar sutures must be tied before the alar cartilages are modeled.

#### MODELING OF THE NASAL TIP

The method for modeling the alar cartilages that has given the best results in my own experience is one originated by Joseph Safian<sup>5</sup> of New York City. The technique of the "Safian tip," as I like to call it, has, as far as I know, been nowhere described in detail. For that reason I am bringing it with Dr. Safian's approval, to the attention of rhinoplastic surgeons. The success of this technique, which I pre-

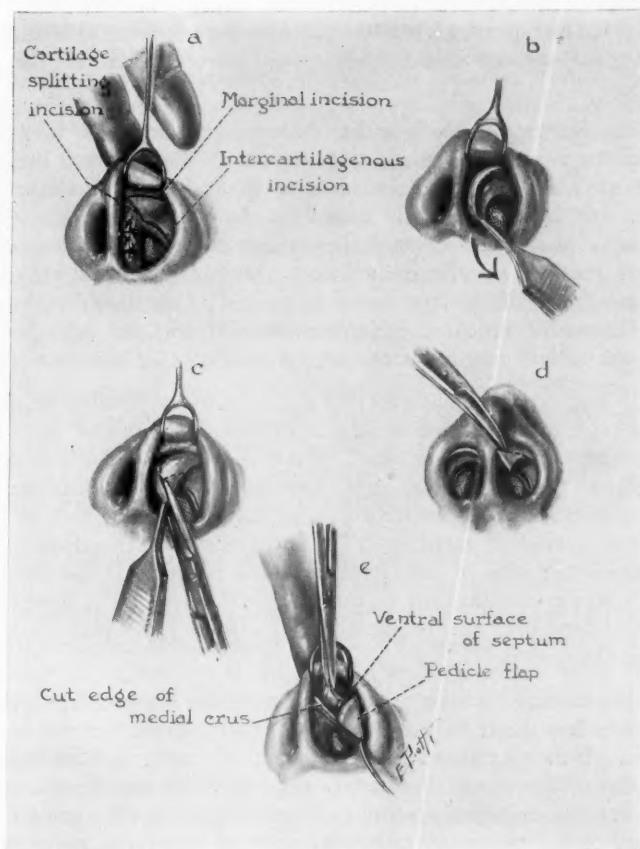


Fig. 2.—Modeling of the Alar Tip Cartilages.

a. Columella sewed to septum *before* modeling of alar tip cartilages. Three incisions bounding pedicle flap, which remains attached laterally for blood supply.

b. Pedicle flap made up of lateral crus and underlying vestibular skin dissected thoroughly from overlying skin and *rotated* outward beyond rim of nostril.

c. Narrow crescent of cartilage and attached vestibular skin removed from medial portion of flap.

d. Excision of triangular section of lower part of lateral crus which extends beyond rim of nostril. Right side has been similarly treated.

e. Fibrous tissue underlying skin of nasal tip in some cases, contributing to deformity, removed with forceps (not shown) and scissors. Pedicle flap has been temporarily pulled out of position to facilitate removal.

sent here with very minor modifications, depends on how the lateral crus of the alar cartilages is dissected from the overlying skin through a marginal incision and a cartilage-splitting incision of the alar cartilage.

The marginal incision is the one usually recommended by most plastic surgeons. It is made with a No. 15 Bard-Parker blade or small, curved double-edge Joseph scalpel just inside the rim of the nostril and is begun slightly medial to the angle of the tip, where the medial and lateral crura of the alar cartilages meet. The incision is then carried laterally immediately anterior to the lower border of the cartilage of the lateral crus to the ala. One should make sure that the medial end of the marginal incision does not extend down onto the medial crus but stops high in the apex of the nasal tip.

If one carefully observes the tip, it is often possible to detect at the angle of the alar cartilage a small web or triangle of outer skin and underlying vestibular skin. The base of this triangle is at the margin of the nostril, the apex directed upward. In making the marginal incision, one must be careful not to mar this web of skin, which is devoid of cartilage. The incision should therefore closely hug the lower edge of the lateral crus and be made as far from the nostril rim as the anatomy in each case will allow. The lower edge of the lateral crus can be identified as a faint ridge just inside the rim of the nostril.

The cartilage-splitting incision through the alar cartilage is much the more important of the two incisions and determines the artistic success of the operation. It must be made with meticulous care. The rim of the nostril is everted, using a two-pronged ball-end retractor. The middle finger of the hand placed on the external skin surface of the tip exerts counterpressure to bring the angle of the alar cartilage conveniently into the operative field. With a No. 15 Bard-Parker blade, the incision is actually made on the medial crura high in the apex, medial to the angle of the alar cartilage, beginning at the medial end of the previously made marginal incision. The incision is carried in a dorsal and cephalic direction through the cartilage of the medial crura and membranous septum to the edge of the septum, where its ventral and caudal surfaces meet. The direction of the cartilage-splitting incision, with the patient supine, is a prolongation of the new profile line. The incision appears, however, much more vertical when it is made in the manner described above, owing to the temporary distortion of the medial crus by the counterpressure exerted on the outer skin. This counterpressure by the finger is necessary for bringing the area more clearly into the operative field. When the pressure is released, the ventral surfaces of the

nasal bones, the dorsum of the septal cartilage and the cut edges of the medial crura will lie in the same profile line.

With scissors introduced through the marginal incision, the lateral crus is now dissected free of the overlying skin. The scissors should be applied directly against the cartilage until they reach the initially made intercartilaginous incision. The area bounded by the three incisions—marginal, cartilage-splitting and intercartilaginous—is thus transformed into a pedicle made up of the lateral crus of the alar cartilage and its underlying attached vestibular skin. Through the pedicle's base, which is situated laterally, an adequate blood supply is maintained. The pedicle, whose base is at its lateral aspect, must be dissected from its overlying skin far laterally, especially at its upper border, thus allowing rotation or sliding of the flap forward and outward beyond the rim of the nostril. This maneuver is important in that the rotation of the flap changes the position or direction of the lateral crus. Included in the flap, which is now ready for modeling, is the dome—the hollowed-out portion of the angle of the tip responsible for the bulbous, unattractive nose.

The pedicle flap is now ready for trimming. It should be grasped with fine-tooth forceps and gently rotated outward beyond the rim of the nostril. No undue tension on the flap need be exerted. By this maneuver the thoroughly dissected and freely movable upper outer border of the alar cartilage assumes a somewhat more medial position. A narrow ellipse or crescent of the medial border of the flap, together with the underlying vestibular skin, is then removed with suitable, delicate scissors. The amount to be removed—usually about an eighth of an inch wide—depends on the breadth of the tip and will be determined by the skill and judgment of the surgeon. If desired, more of the vestibular skin may be retained and the cartilage alone excised. Atresia of the nostril does not follow when the ellipse of cartilage with its underlying vestibular skin is removed, provided of course that the surgeon observes a reasonable degree of caution and is judicious in the amount of cartilage he excises.

There now remains to be resected the lower or caudal edge of the rotated lateral crus of the alar cartilage which extends outside the nose beyond the rim of the nostril. The resected portion is usually in the form of a triangle, with its base medially and its apex laterally placed. The idea of resecting the caudal edge of the lateral crus may come as a shock to surgeons who have gone on the principle that under no circumstances should the lower edge of the lateral crus of the ala be excised. But it is precisely this procedure that makes the difference between a highly satisfactory result and a mediocre

one. The excised cartilage with its underlying vestibular skin extending outside and beyond the rim of the nostril is resected with delicate, straight scissors directly across in line with the margin of the nostril. The amount resected depends on the size of the alar cartilages and on the amount of cartilage that extends beyond the marginal incision after the flap has been rotated outward. But first, the surgeon should inspect the vestibule to make sure that the upper edge of the alar cartilage approximates and touches that part of the upper lateral cartilage which he has left behind. Care should be taken when the alar cartilage is rotated forward not to remove too much of its lower border.

By changing and altering the size, shape and direction of the alar cartilages, the surgeon can transform the broad, bulbous, drooping tip so completely that the nose becomes a beautifully modeled, attractive feature. It is not enough, however, merely to reduce the size of the alar cartilages, leaving the original shape and position unchanged. One must realize that in the correction of the congenital convex dorsum, the ventral surfaces of the alar cartilages are usually a continuation of the undesirable convexity of the dorsum of the entire nose. The alar cartilages are themselves convex. A change in their size, shape and position is therefore necessary. All incisions, underminings and excisions on the alar cartilages must be of equal size and length, to ensure symmetry of the tip. After the cartilage-splitting incision is made, the medial crura need no further shaping or trimming.

In some individuals, a considerable amount of redundant fibrous tissue can be found under the external skin of the nasal tip, usually between the medial crura just above the very apex of the nasal tip on its ventral border. In cases where it contributes, as it sometimes does, to the deformity of the tip, it should be removed. The fibrous tissue can be grasped with tooth forceps after exposure with a double ball-point retractor at the rim of the nostril and removed with scissors held parallel with the outer skin. Care should be exercised to avoid injuring the skin.

At the operation's end, the medial crura are held attached to the nasal septum by the previously placed septocolumellar sutures. Their ventral surfaces form a prolongation of the new profile line and constitute the apex of the new tip. The lateral crura in their new position are in an immediately adjoining position laterally but never overlap the medial crura at the tip's apex. The ventral portion of the medial crus just under the apex of the tip is on a higher level than that part of the medial crus immediately adjoining the nasal septum. The tip is thus given a slight uptilt.

At no time is it necessary or even desirable to detach or separate the outer skin of the columella from the underlying cartilage. The lateral crura merely approximate each other at the dorsum of the nose in midline just above the apex of the tip. They should never override each other at midline. Appropriate strips of adhesive tape hold the nasal bones and alar cartilages in place; they can be removed after five days. The black silk suture at the septocolumellar incision should be removed in ten days. No packing whatsoever need be used inside the nose following the operation.

The Safian clamp is highly satisfactory for holding the nasal bones in place and should be applied as soon as the operation is over. No sloughing of skin will occur if the clamp exerts no more than light finger pressure, nor will the blood supply to the skin be interfered with. The use of this clamp also prevents the formation of hematoma between the skin and nasal bones, keeps postoperative swelling down to a minimum and gives a narrow nose. After 48 hours, when the patient is ready to be discharged from the hospital, the clamp may be removed.

#### SUMMARY

The *invaginating technique* in cosmetic rhinoplasty is described in detail. Its purpose is to maintain the newly modeled nasal tip in the position given it by means of rhinoplasty by preventing secondary dropping.

In addition the technique perfected by Safian in the modeling of the tip is described.

520 BEACON STREET.

#### REFERENCES

1. Joseph, J.: Nasenplastik und Santige Gesichtsplastik, C. Kahtzck, Leipzig, 1931.
2. Daley, Jacob: Role of Columellar and Septo-columellar Sutures in Rhinoplasty, Arch. Otolaryng. 45:178-190 (Feb.) 1947.
3. Fomon, Samuel, Gilbert, J. G., Silver, A. G., and Syracuse, V. R.: Plastic Repair of the Obstructing Nasal Septum, Arch. Otolaryng. 47:7 (Jan.) 1948.
4. Cottle, M. H., and Loring, R. M.: Corrective Surgery of the External Nasal Pyramid and the Nasal Septum for Restoration of Normal Physiology, Illinois M. J. 90:119-131 (Aug.) 1946.
5. Safian, Joseph: Corrective Rhinoplastic Surgery, New York, The American Journal of Surgery, Inc., 1935.

## THE DIAGNOSIS AND TREATMENT OF TUMORS OF THE NECK

DELMAR F. WEAVER, M.D.

DETROIT, MICH.

The purpose of this paper is to consider the management of patients with tumors of the neck. Inflammatory and cystic tumors as well as neoplasms are included, while primary tumors of the larynx, pharynx and esophagus are not.

Tumors of the skin and subcutaneous tissue, with few exceptions, are not grave. Nevi are removed when unsightly or subject to irritation by clothing. Those with a deep, broad base are usually excised while those with a narrow base or pedicle are electrocoagulated.

Keloids are excised and the wound irradiated soon after healing.

Epitheliomas of the skin are excised with a margin of normal tissue. The width of the margin is determined, to some extent, by the degree of the malignancy. The highly malignant melano-epitheliomas should be removed with a wide margin, even though from the standpoint of a cure this is apt to be useless. Webster, Stevenson and Stout<sup>1</sup> reported a study of 162 of these tumors, 9 of which were in the neck. They consider excision superior to electrocoagulation because of slow healing, conspicuous scarring and lack of a specimen for examination following the latter. This decision is no doubt wise in the neck where there is ample skin to permit a wide excision and to obtain a satisfactory closure.

Irradiation is ineffectual.

Erich<sup>2</sup> has given an interesting discussion of sebaceous, mucous, dermoid and epidermoid cysts. He points out that sebaceous cysts tend to become infected and drain, and that occasionally cancer develops in them. New and Erich<sup>3</sup> reported 66 dermoid cysts of the head and 37 of the neck. Since the removal of these tumors, as well as fibromas, lipomas and neurofibromas, is usually by simple excision, they are interesting chiefly from the standpoint of differential diag-

---

From the Division of Otolaryngology, Henry Ford Hospital, Detroit, Michigan.

Read before the meeting of the Ear, Nose and Throat Section of the American Medical Association, Atlantic City, N. J., June 9, 1948.

nosis. These tumors are usually superficial and movable and do not give the impression of having a deep attachment. Fibrolipomas are quite vascular, as a rule, and frequently occur in the suboccipital region.

I recently removed a small superficial draining sinus from low on the neck of a little girl. The fluid draining from it was thin and clear. Microscopic examination disclosed the presence of salivary gland tissue.

When the diagnosis of dermatological conditions of the neck is not definite, a specimen of tissue should be removed for microscopic examination unless the lesion is small, in which case it should be completely excised.

Radiodermatitis, following irradiation of the neck, is prone to cause epithelioma and consequently should be removed. When superficial and small the areas can be excised. When larger, it is necessary to apply a free skin graft after removal and when the deeper tissue is involved a pedicled flap may be necessary.

Tumors of the blood and lymphatic vessels usually involve the skin and they frequently extend to deeper tissues. Watson and McCarthy<sup>4</sup> reported a study of 1308 hemangiomas, 4% of which were in the neck.

Capillary hemangiomas are superficial and are important chiefly from a cosmetic standpoint. If conspicuous and the patient wishes their removal, they should be excised. If large in size, excision in stages may be necessary.

Cavernous hemangiomas extend to the subcutaneous tissue and muscle, and may be mixed with the capillary type. They may become connected with the general arterial system so that they pulsate and constitute a potential danger from hemorrhage. Figi<sup>5</sup> mentions the advisability of ligating the afferent and efferent vessels before treating certain of these tumors.

Hemangiomas are compressible and the overlying skin is usually involved to some degree. In infancy and early childhood these tumors are radiosensitive, but this tendency decreases as the child grows older. Consequently, while spontaneous recovery may occur, delay in treatment is not advisable. Whether irradiation is carried out by roentgen therapy, radium element or radon seeds depends upon the availability of material, the location of the tumor and the experience of the operator. If the skin is rather thoroughly involved by the tumor, roentgen rays may have an advantage. If the tumor is deep and covered by relatively normal skin the insertion of gold radon seeds has a distinct advantage.

Radioresistant tumors in older patients that cannot be excised with safety, are best treated by the injection of sclerosing materials. Sodium morrhuate is one of the suitable materials for this purpose. It is important to consider all methods of treatment and to employ the method or combination of methods that is most suitable for the particular characteristics of each case. In the presence of vascular tumors in the neck, aneurysms should be thought of.

Lymphangiomas are treated in much the same manner as the hemangiomas. The type known as the cystic hygroma usually occurs in the neck. These tumors may be quite extensive and at times extend into the mediastinum. Figi<sup>6</sup> feels that there is a distinct advantage in irradiating them before surgical removal.

The malignant tumors of endothelial origin are rare. They tend to respond to irradiation.

Thyroglossal duct cysts and branchial cleft cysts and sinuses are of developmental origin and although promptly suspected, due to their characteristic history, location and appearance, their accurate pre-operative diagnosis is frequently difficult because of their resemblance to other tumors that happen to be in the same location. The thyroglossal duct cysts are likely to be confused with dermoid cysts and thyroid adenomas while the branchial cleft cysts may be mistaken for lymphadenopathy or confused with the unusual lateral aberrant thyroids and carotid body tumors.

Thyroglossal duct cysts may have been apparent for a number of years or may have made a recent appearance. There is frequently a history of infection followed by incision and drainage. They seem to be attached to the hyoid bone and move with it on swallowing. Although a large number of them have no doubt been successfully removed by simple excision without disturbing the hyoid bone, it appears that all recurrences are in such cases or in those cases in which the hyoid bone has been divided, but the duct from the hyoid bone to the foramen caecum has not been removed. Apparently the only operation that assures a satisfactory result is the often mentioned procedure described by Sistrunk.<sup>7</sup> A transverse incision about 3 in. long is made over the cyst and it is dissected to the hyoid bone. A section 1 or 2 cm wide is removed from the body of the hyoid bone and the muscle of the tongue is cored out to the foramen caecum. The latter procedure is facilitated by the operator's left index finger being placed in the patient's mouth and pressed forward and upward on the foramen caecum. Although it is advisable to clamp and tie the stump of the muscle and duct at the foramen caecum, it does not seem necessary to enter the oral cavity. The divided ends of the

hyoid bone should be approximated by a mattress suture of catgut. Drainage is not always necessary although a soft rubber drainage tube may be inserted for a few days.

Some idea of the frequency with which these cysts occur can be gained from Lahey's report<sup>8</sup> that 314 of these cysts had been removed at the Lahey Clinic by 1947. Twenty-seven branchial cleft cysts and sinuses had been removed at the same institution in a ten-year period.<sup>9</sup>

Branchial cleft cysts and sinuses may have been present a long time before the patient seeks treatment and there may be a history of acute infection in them. The tumor lies anterior to the sternomastoid muscle and appears to be superficial.

For removal of a branchial cleft cyst an incision is made through the skin and subcutaneous tissue, over the cyst, and in the direction of the lines of the neck. The skin is then undermined so that the fascia can be incised along the anterior border of the sternomastoid muscle. The dissection must then be carried out carefully for it is usually desirable to preserve the cyst intact during the early part of the dissection. During the latter part the dissection is close to important vessels and nerves. The dissection is carried to the mucous membrane of the pharynx and the stump usually clamped and tied, but it does not seem necessary to invert the stump or enter the pharynx.

In removing a sinus which opens low on the neck several short transverse incisions at successive levels result in less scarring than a vertical incision.

The report by Neel and Pemberton<sup>10</sup> based on a study of 319 cases is the largest group with which I am familiar.

Involvement of the submaxillary salivary glands by mumps or acute mononucleosis occurs but there are usually associated signs, symptoms and history of a general nature which help in making the correct diagnosis. When chronic enlargement occurs in one gland, the diagnosis may be more difficult. In general, if the possibility of a stone in the gland or at the junction of the gland and duct exists, x-ray examination of the floor of the mouth should be done. If a stone is present its removal through the mouth is indicated unless there is sufficient chronic infection in the gland to make removal of the gland advisable.

The commonest type of neoplasm of the submaxillary gland is the mixed tumor and complete removal of the gland should suffice, for cure. The cylindroma type of tumor, however, is more malig-

nant and has infiltrative tendencies. Dockerty and Mayo<sup>11</sup> advise not only wide removal of the gland with Stenson's duct and adjacent suspicious tissue, but also dissection of the cervical lymph nodes in most cases.

Lahey<sup>8</sup> refers to the occasional occurrence of a sublingual thyroid gland and warns that complete removal will result in complete myxedema. He also states that all discrete adenomas of the thyroid gland should be removed because of the danger of malignant degeneration. He mentions the high incidence of cancer in the unusual lateral aberrant thyroid tissue.

Actinomycosis is a slowly developing inflammatory process. The diagnosis is made by finding the sulfur granules in pus from a freshly opened abscess.

Involvement of the cervical lymph nodes by syphilis is usually associated with involvement of the mucous membrane of the air or food passages.

Carotid body tumors are located deeply and tend to enlarge upward and inward toward the pharynx. Harrington, Clagett and Dockerty<sup>12</sup> advise complete surgical removal, with ligation of the carotid vessels being necessary in about 50% of the cases. Lahey,<sup>8</sup> however, reserves removal for the cases in which it is possible to completely preserve the integrity of the carotid vessels.

Correct evaluation of cervical lymphadenopathy is difficult. The size, consistency and degree of tenderness of the glands vary in patients and also in the same patient from time to time. The shape, conformation and position of the neck, as well as the age of the patient, are also factors which must be taken into consideration.

The discovery of a firm, enlarged cervical lymph gland which is nontender should indicate the possibility of serious disease until there is evidence to the contrary. A careful otolaryngological examination should be performed. If a primary malignant tumor is found it can usually be taken for granted that the gland is secondary to it. If the nose and throat examination is negative and no significant findings result from general physical examination and laboratory studies, the gland should, in most cases, be removed for microscopic examination.

Primary involvement of the lymph glands may be due to lymphoblastoma, either Hodgkin's type or lymphosarcoma, and roentgen ray treatment is usually indicated.

If metastatic carcinoma is present the location of the primary tumor and the microscopic grading of the tumor (Broders) are im-

portant in choosing a method of treatment. If the tumor is highly malignant (Grade IV), as are most of the cancers of the nasopharynx, irradiation is the most effective method of treatment. If the tumor is moderately malignant (Grade II and III) dissection of the cervical lymph glands offers the best chance of cure. Blair, Moore and Byars,<sup>13</sup> Figi,<sup>14</sup> and Frazelle<sup>15</sup> recommend block dissection of the cervical glands on the side of the lesion in most cases of cancer of the tongue, floor of the mouth or cheeks. The superficial glands, consisting of the submental, submaxillary and upper deep cervical, on the opposite side are frequently removed at the same time. Although Erich<sup>16</sup> advises bilateral superficial cervical gland dissection routinely in carcinoma of the lower lip other operators advise operation only after the glands have become clinically involved. They then recommend a block dissection on the involved side with or without removal of the superficial group on the opposite side. When the location of the primary tumor is such that inaccessible lymphatic glands are apt to be involved, removal of the cervical glands is not advisable.

I have recently treated a patient who had an epithelioma involving the left anterior tonsillar pillar by electrocauterization of the local lesion followed by block dissection of the lymphatic glands of the left side.

Another patient had an epithelioma involving the anterior end of the nasal septum and columella which was treated by thorough electrocauterization of the local lesion followed by removal of the submental lymph glands and the submaxillary and upper deep cervical glands on both sides.

If there is reasonably good evidence that tuberculosis has invaded the glands, such as evidence of tuberculosis in some other part of the body or the characteristic matting together and inflammatory appearance of the glands, the gland should not be removed. Usually roentgen therapy is quite valuable in the treatment of tuberculous lymphadenopathy unless the gland is draining, in which case the gland should be removed. Streptomycin is of value.

Involvement of the cervical lymph glands by Boeck's sarcoid is seen occasionally and responds readily to proper doses of roentgen ray therapy. I recently treated a patient who had a large retropharyngeal tumor associated with cervical lymphadenopathy. Microscopic examination of a cervical lymph gland revealed the presence of Boeck's sarcoid. Roentgen ray therapy brought about complete recovery of the local lesion.

The diagnosis of acute mononucleosis is confirmed by the heterophil agglutination test.

There is a small group of adults who have a generalized chronic lymphadenopathy from no known cause and with no apparent ill effects.

Most operations on the neck can be carried out with local infiltration or cervical block anesthesia. Novocain is used for this. General anesthesia is preferable for dissection of lymph glands since it is necessary to inject the floor of the mouth when cervical block is used. General anesthesia is preferable for the removal of thyroglossal duct cysts also.

#### HENRY FORD HOSPITAL.

#### REFERENCES

1. Webster, Jerome P., Stevenson, Thomas, and Stout, Arthur Purdy: The Surgical Treatment of Malignant Melanomas of the Skin, *Surg. Clin. North America* 24:319-339 (Apr.) 1944.
2. Erich, John B.: Sebaceous, Mucous, Dermoid and Epidermoid Cysts, *Am. J. Surg.* 50:672-677 (Dec.) 1940.
3. New, Gordon B., and Erich, John B.: Dermoid Cysts of the Head and Neck, *Surg. Gyn. and Obst.* 65:48-55 (July) 1937.
4. Watson, W. L., and McCarthy, W. D.: Blood and Lymph Vessel Tumors: 1056 Cases, *Surg. Gyn. and Obst.* 71:569-588 (Nov.) 1940.
5. Figi, Frederick A.: Treatment of Angioma of the Face, *Arch. Otolaryng.* 24:271-281 (Sept.) 1936.
6. Figi, Frederick A.: Radium in the Treatment of Multilocular Lymph Cysts of the Neck in Children, *Am. J. Roentgenol. and Radium Therap.* 21:473-480 (May) 1929.
7. Sistrunk, Walter E.: Technique of Removal of Cysts and Sinuses of the Thyroglossal Duct, *Surg. Gyn. and Obst.* 46:109-112 (Jan.) 1928.
8. Lahey, Frank H.: Tumors of the Neck, *Surg. Clin. North America* 27: 486-500 (June) 1947.
9. Lahey, Frank H., and Nelson, Harlan F.: Branchial Cysts and Sinuses, *Ann. Surg.* 113:508-512 (Jan.-June) 1941.
10. Neel, Harry B., and Pemberton, John de J.: Lateral Cervical (Branchial) Cysts and Fistulas, *Surgery* 18:267-286 (Sept.) 1945.
11. Dockerty, M. B., and Mayo, C. W.: Primary Malignant Tumors of the Submaxillary Gland with Special Reference to Mixed Tumors, *Proc. Staff Meet. Mayo Clin.* 17:593-603 (Dec. 16) 1942.
12. Harrington, S. W., Clagett, O. T., and Dockerty, M. B.: Tumors of Carotid Body, *Ann. Surg.* 114:820-833 (Nov.) 1941.
13. Blair, Vilray P., Moore, Sherwood, and Byars, Louis T.: Cancer of the Face and Mouth, St. Louis, C. V. Mosby Co., 1941.
14. Figi, Frederick A.: Malignancy of the Upper Respiratory Tract and Adjacent Structures, *Surg. Gyn. and Obst.* 62:498-502 (Feb.) 1936.
15. Frazell, E. L.: Personal communication.
16. Erich, John B.: Treatment of Carcinoma of the Lips, *Surg. Clin. North America* 27:995-1006 (Aug.) 1947.

## XXI

### BACKSTAGE WITH THE BOARD

ARTHUR W. PROETZ, M.D.

ST. LOUIS, MO.

May, of this year, will mark the twenty-fifth anniversary of the American Board of Otolaryngology. To recall at this time the struggles attending its birth and childhood, the internecine turmoils of its adolescence and all the sweat, blood and tears shed along the wayside by its members would be of small interest to any except those members. To recount briefly how and why it came into being, how it functions and conducts its examinations, and how it keeps in tune with the progressive spirit of the time may divert those who have had dealings with it in the past and give some measure of comfort to the candidates of the future.

In the early years of the present century training in otolaryngology was a very personal thing and as such a very uncertain one. A candidate for the specialty either attached himself to a preceptor or, to put it less elegantly, worked in a doctor's office; or, if funds were available he went to Europe, either making the grand tour of the capitals, picking up what he could from the masters, or settling in some center, preferably Vienna, where the teaching was highly concentrated and highly specialized.

Under a preceptor his training was sharply circumscribed by the interests and the limitations of that practitioner. If he went to Vienna his curriculum was apt to be channeled into a pattern of his own inexperienced devising. He picked his teachers for himself and paid them individually for their time and talents. In either case it was a strenuous career requiring more than average time, funds and effort.

Then came the first World War. With the possible exception of Ethelred the Unready no one was ever less prepared than our Uncle Samuel, at least in so far as his medical specialists were concerned. A first lieutenant in the Medical Reserves with a natural leaning, let us say, toward urology was suddenly snatched up by his new Sam Browne belt, projected into some new camp hospital and by a special Order of the Day annointed an otolaryngologist. His training consisted of several weeks' contact with a likewise newly-annointed

major recently chosen from the better strata of civilian otolaryngologists.

With the exigencies of the time, he was then whisked off to France, a copy of Ballenger under his arm, to pry shrapnel fragments out of ethmoids and petrous pyramids, not to mention more remote and unrelated structures.

By the end of the war and a 'flu epidemic or two, he had picked up a rough working knowledge of otolaryngology with a military flavor and was ready to hang up his shingle as such in the Medical Arts Building at home. It was the low point in the history of otolaryngology in America where this young specialty had already achieved distinction.

The need was urgent for some type of evaluating agency to supplement the general licensing regulations of the various State Boards of Health. In May 1917, the three national ophthalmological bodies had established the American Board of Ophthalmic Examinations which later became the American Board of Ophthalmology. The American Academy of Ophthalmology and Otolaryngology had also for some time established a small committee for screening applicants to membership by means of an oral, didactic examination.

In 1924, the American Otological Society, the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology, and the Section on Otolaryngology of the American Medical Association each appointed two members to form a committee for the organization of an examining board. This committee met on November 10th of that year at the University Club in Chicago and discussed the formation of the American Board of Otolaryngology. Dr. George E. Shambaugh, who was not a member of the committee, was invited to be present as a guest and to serve as temporary chairman until the assembly could adopt a constitution and by-laws and elect officers.

Lists of members of the component societies were prepared and it was decided that those receiving the unanimous approval of the Board should be certified without examination. For a number of years it was the practice of the Board to grant such formal certification to candidates of high professional standing, who were obviously entitled to it and who had the unanimous approval of the examiners. The practice was discontinued in 1942 as the need for it no longer existed.

The Board quickly earned the support and approval of most of the outstanding specialists and educators, although there was oppo-



JOSEPH C. BECK†



LAWRENCE R. BOIES



THOMAS E. CARMODY†

sition in some quarters as might have been expected: "What right have these . . . etc." But the Board had no wish to set itself up as either a judge or a censor of its contemporaries and took pains never to appear as though it had. Obviously all of the candidates were not competent; someone had to decide; members of the Board were not acting individually but as the chosen representatives of all of organized otolaryngology in America. It was too much to expect that those whom the Board was organized to discourage would fall in with the idea.

For some years the Board contented itself with the routine business of examinations, not of candidates alone, but of itself as well, striving for a detached and yet sympathetic approach eliminating personal prejudices. Gradually it became apparent that there was a general weakness exhibited by the candidates in certain branches of

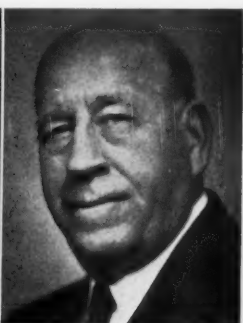
LOUIS H. CLERF

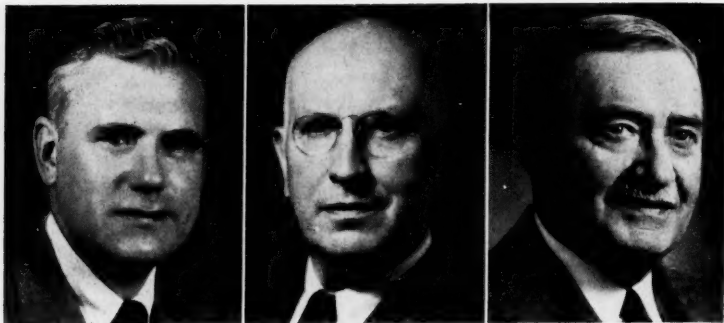


GEORGE M. COATES



LEE WALLACE DEAN†





RUSSELL M. DECKER

RALPH A. FENTON

PERRY G. GOLDSMITH

the specialty for the simple reason that instruction in these branches was not to be had or was available to a very few.

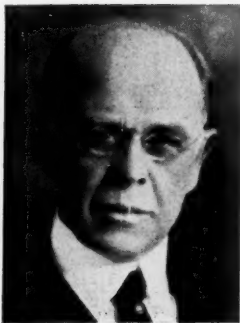
In the beginning, low grades in pathology had largely to be ignored because candidates, otherwise competent, threw up their hands in dismay when confronted with a microscope. It had never occurred to them to look at their own specimens and they would not have understood them had they done so. Examinations in microscopy continued, however, and when for a few years candidates were conditioned in pathology, medical schools responded by supplying the much needed instruction in that branch. Later physiology underwent the same process. Even within the Board there was some opposition to questions in physiology on the ground that they were abstract if not abstruse and had little to do with the competent practice of the specialty. But not for long, for obviously it is as im-

WILLIAM E. GROVE

THOMAS H. HALSTED

GORDON F. HARKNESS

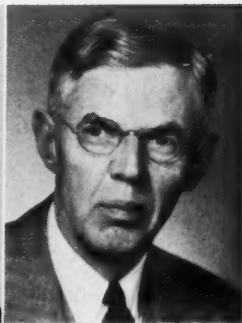




THOMAS JEFFERSON HARRIS†



LYMAN HEINE



FREDERICK T. HILL

portant for the practitioner to know how his machine works as how it is put together.

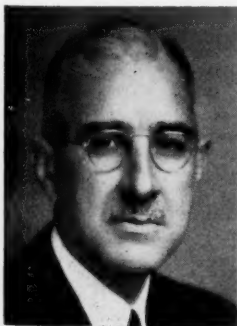
The president and the secretary function only as executives. Even the slightest problems of policy are determined by the Board as a whole. At each meeting one full session is devoted to introspection. The Board examines the Board to improve examinations, to minimize inequalities and to spare the candidate unnecessary hardship. For a time the candidate was known to the examiner only by a number. This carried impersonality to the verge of chilliness and was abandoned although it is still considered bad taste to ask a candidate where he comes from and who his friends are.

It may interest future applicants to know how their applications are processed and how the examinations are conducted:

GORDON D. HOOPLE

WESTLEY M. HUNT

PERCY E. IRELAND

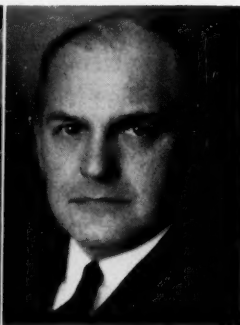




MARVIN F. JONES



DEAN M. LIERLE



HAROLD I. LILLIE

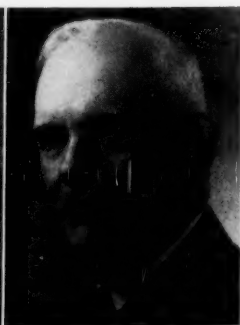
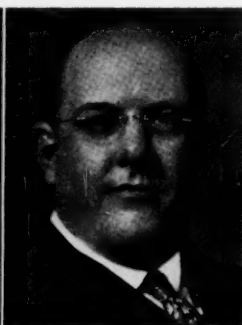
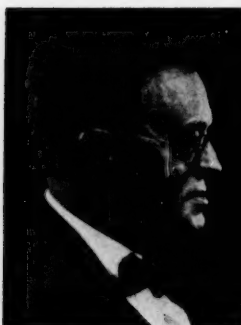
On receipt of an application it is first examined to determine whether the requirements as outlined in the current leaflet are satisfied. This applies to general and special training. It is the policy of the Board to refer questions of morals and ethics to the local county societies. The province of the Board is professional evaluation and not censorship. Comments on the fitness of the candidate are next requested by mail both from sources suggested by the applicant and from other reliable sources in his community.

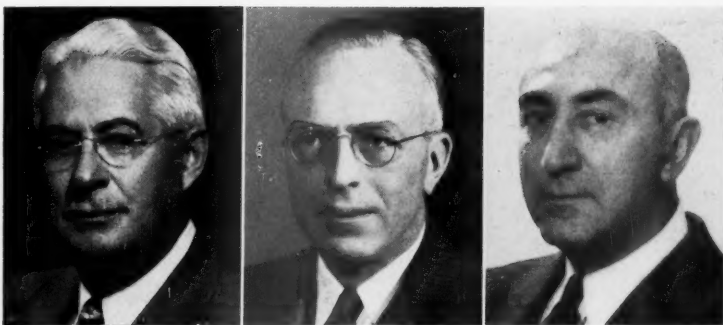
Candidates are then given appointments for examination in order of application up to the number which can be satisfactorily managed at each examination. This depends somewhat upon the local hotel and hospital facilities. On an average of 70 can be accommodated without curtailing the time and materials to which the candidate is entitled. During the war years the number once reached

HANAU WOLFF LOEB†

ROBERT CLYDE LYNCH†

CARL H. McCASKEY





BERNARD J. McMAHON

JAMES H. MAXWELL

PHILIP E. MELTZER

149, but such numbers create difficult situations for both candidate and examiner. The smaller number assures an abundance of clinical material and microscopes, so important to a comprehensive examination.

Examiners are constantly being reminded that they are not there to pick out an army of superspecialists but that they are examining the next generation of their fellow practitioners for competence in the generally accepted meaning of the term. In grading the applicant they ask themselves, "Could one conscientiously refer a patient to this doctor?"

The candidate meets four examiners in turn on didactic subjects and theory. Great care is taken to cover the combined subjects of the ear, nose and throat but at the same time some overlapping is

HARRIS P. MOSHER

WERNER MUELLER

WILLIAM VALENTINE MULLIN†





C. STEWART NASH

ARTHUR W. PROETZ

ROBERT F. RIDPATH

provided so that a candidate who fails to distinguish himself with one examiner may retrieve his fortunes with another.

An examination in pathology, general and microscopic, is now required of all candidates.

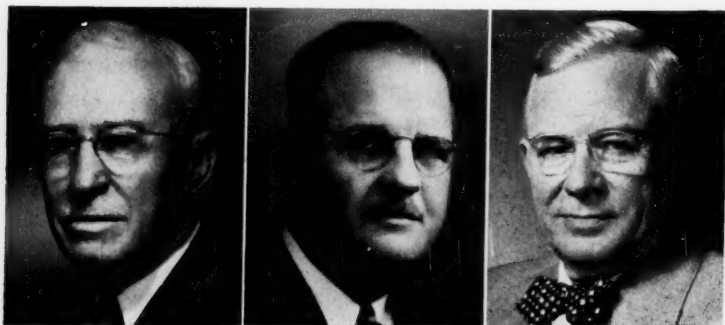
On the second day the candidate examines three patients, each under the eye of a different examiner who grades the history, the examination and the subsequent discussion of the case. All laboratory information—x-rays and laboratory reports—and supplementary instruments such as tuning forks and transillumination equipment, are available to the candidate but only if he asks for them specifically. In the final grading much importance is attached to the details of the clinical phases.

The third day is occupied with discussion and grading of candidates. The Board is not interested in grades as such, the vital con-

W. E. SAUER

LEROY A. SCHALL

ERNEST M. SEYDELL





JOHN J. SHEA



BURT R. SHURLY



ROSS HALL SKILLERN†

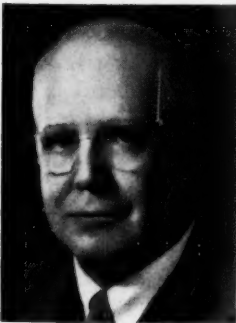
sideration being, of course, "Does this man merit a certificate?" Impersonality prevails and there is every safeguard against prejudice.

During the discussion of a candidate his portrait is projected on a screen so that there shall be no confusion as to his identity. Then each examiner in turn reads his grade aloud. If the candidate obviously passes, that ends it. If he has a failing grade or several on the borderline, the questions and answers are read out and discussed by the whole Board. Finally it is not only the examiners before whom he has appeared, but the whole Board which decides his certification, condition or failure. Suspicion occasionally entertained by unsuccessful candidates that a certain examiner "flunked" them is without foundation. At least half the Board and the candidate himself have been party to it.

FRANK R. SPENCER

O. E. VAN ALYEA

WILLIAM P. WHERRY†





FLETCHER D. WOODWARD

It takes several years' experience to produce a good examiner. It is not always easy on the one hand to distinguish knowledge from fast conversation, semantics and a confident air, or on the other to make reasonable allowance for fright or a bad night on the train. Nevertheless guest observers who sometimes attend these grading sessions are surprised at the uniformity of the grades of a given candidate. It is not unusual to encounter a divergence of only three or four per cent among the eight grades.

The accumulated results of these examinations over a period of years have thrown some useful sidelights on the teaching of otolaryngology in American medical schools and hospitals which are detectable through no other agency.

Information so gathered has always been available to the heads of departments and hospitals for the improvement of their teaching facilities. Last year and the year before, on the day preceding the meeting of the Academy, the Board has invited the heads of the departments of otolaryngology of all teaching institutions, including hospitals, to meet with it informally for the purpose of discussing common problems. The discussions at these meetings are recorded but are not published. Freedom of expression is thus encouraged and by general request these meetings will be continued.

Two examiners meet regularly with the Advisory Board for Medical Specialties as members of that body. This Board acts, as its name indicates, solely in an advisory capacity but it assures among the various special boards some measure of uniformity in meeting the ever changing requirements.

These are crucial times for American medicine. What more can one say for a body such as this, on its Silver Jubilee, than that

it has always been and always will be dedicated to the highest ideals and a tireless devotion to the public weal?

1010 BEAUMONT BLDG.

## THE AMERICAN BOARD OF OTOLARYNGOLOGY

### HAS HELD THE FOLLOWING EXAMINATIONS:

- |  |   |
|--|---|
| 1925—Philadelphia, Pa.<br>Chicago, Ill.  | 1936—Kansas City, Mo.<br>New York City, N. Y.   |
| 1926—Dallas, Texas<br>San Francisco, Calif.<br>Denver, Colo.<br>Montreal, Canada | 1937—Atlantic City, N. J.<br>Chicago, Ill.      |
| 1927—Washington, D. C.<br>Spokane, Wash.<br>Detroit, Mich.<br>Memphis, Tenn.     | 1938—San Francisco, Calif.<br>Washington, D. C. |
| 1928—Minneapolis, Minn.<br>New York City, N. Y.<br>St. Louis, Mo.                | 1939—Chicago, Ill.<br>Chicago, Ill.             |
| 1929—Portland, Ore.<br>Philadelphia, Pa.   | 1940—New York City, N. Y.<br>Cleveland, Ohio    |
| 1930—Detroit, Mich.<br>Chicago, Ill.   | 1941—Cleveland, Ohio<br>Chicago, Ill.           |
| 1931—Los Angeles, Calif.<br>Philadelphia, Pa.<br>Indianapolis, Ind.              | 1942—Philadelphia, Pa.<br>Chicago, Ill.         |
| 1932—New Orleans, La.<br>Montreal, Canada  | 1943—New York City, N. Y.<br>Chicago, Ill.      |
| 1933—Chicago, Ill.<br>Boston, Mass.  | 1944—New York City, N. Y.<br>Chicago, Ill.      |
| 1934—Cleveland, Ohio<br>Butte, Mont.<br>Chicago, Ill.<br>San Antonio, Texas      | 1945—New York City, N. Y.                       |
| 1935—New York City, N. Y.<br>Cincinnati, Ohio                                    | 1946—Chicago, Ill.<br>Chicago, Ill.             |
|  | 1947—St. Louis, Mo.<br>Chicago, Ill.            |
|  | 1948—Chicago, Ill.<br>Chicago, Ill.             |
|  | 1949—New York City, N. Y.<br>Chicago, Ill.      |
|  | 1950—New Orleans, La.<br>San Francisco, Calif.  |

# Clinical Notes

XXII

## ACTINOMYCOSIS OF THE PHARYNX AND BASE OF TONGUE

CASE REPORT

WILLIAM WESLEY WILKERSON, JR., M.D.

AND

LEE FARRAR CAYCE, M.D.

NASHVILLE, TENN.

The nomenclature of actinomycosis is nebulous as shown by the lack of unanimity in classification and terminology. There are also differences in opinion as to whether specific fungi are pathogens. This confuses one when reading articles by different authors.

The term "actinomyces" has been proposed by the Committee of the Society of American Bacteriologists for the classification of organisms known in literature by various names, such as, cladothrix, streptothrix, actinomyces, nocardia, copora, actinocladothrix, micromyces and discomyces.

Conant and Rosebury<sup>1</sup> state, "There is no clear evidence that actinomycosis is ever communicable. Actinomycosis bovis is a true parasite of the mucous membrane, never found in nature apart from a parasitic or pathogenic habitat."

Although the literature contains numerous articles describing cases of actinomycosis in which the pathology consists of nodules, necrosis and abscess formation of the tongue, cheek and neck (cervicofacial), one should bear in mind that it does occur in other forms.

Eggston and Wolff,<sup>2</sup> under the heading of "Pharyngitis Sicca," describe a case such as we shall report. They give an excellent brief résumé of this subject.

Zinsser's textbook<sup>3</sup> states, "Standing midway between the true bacteria and the more complex molds there are a number of pathogenic organisms which now have been placed in the order Actinomycetates."

### REPORT OF A CASE

The patient, W. J., aged 17, presented herself, complaining of a constant hacking cough, irritation and fullness of the throat in



Fig. 1.—Clinical appearance of lesion.



Fig. 2.—Clinical appearance of lesion.



Fig. 3.—Photomicrograph showing actinomycetes.

the region of the base of the tongue, which had been annoying her for two weeks. The symptoms were aggravated when the patient was reclining. Upon examination yellow excrescences were noted protruding from many crypts in the base of the tongue and inferiorly behind the epiglottis. Superiorly a few were found on the right lateral pharyngeal band. There was no inflammatory reaction. (Figures 1 and 2 show the distribution and shape of these excrescences.)

The excrescences were rounded in shape, approximately 2 mm in diameter and protruding from the crypts from 1 to 3 mm. The largest one lay anteriorly to the epiglottis.

These yellowish excrescences clung tenaciously to the crypts, yet no bleeding occurred upon removal. Cultures revealed actinomycosis (*buccalis*). The ear, nose and throat and general examinations, including serology, were negative other than some remnants of adenoid tissue.

The excrescences were composed of connective tissue in the center, surrounded by hyperkeratotic epithelium (Fig. 3). Colonies of actinomycosis were found throughout these masses. (This is the same description as given by Eggston.) The treatment consisted of

the daily removal of the excrescences, the painting of the entire area with bismuth violet, roentgen radiation of lymphoid tissue in the area, autogenous vaccine, massive doses of potassium iodide, penicillin aerosol, and 4,200,000 units of penicillin given over a period of four months. With this therapeutic regime, definite improvement was apparent, yet the formation of the irritating masses continued. The infection, however, showed no tendency to spread.

At this point, through the generosity of Dr. E. A. Sharp of Parke, Davis & Company, we obtained a supply of chloromycetin for experimental work. The patient was given 1.5 gm a day. Within a week a marked diminution of both the number and size of the yellowish masses was noted. At the end of three weeks the throat appeared healed. The patient was then given 0.75 gm of chloromycetin a day for a month as a precautionary measure. The other therapy was continued throughout the entire period of treatment, but less frequently after chloromycetin therapy was instituted.

At the end of five months the patient was entirely well and her throat cultures were negative. Her general condition was never involved.

#### CONCLUSIONS

1. The fungi are difficult to classify and their terminology varies.
2. Actinomycosis is the preferable term for the pathogenic fungi in this case.
3. The picture presented is pathognomonic of this type of infection.
4. There are possibly many therapeutic measures of value in such a case.
5. The authors had a similar case several years ago which received the same treatment as the case herein reported, with the exception of chloromycetin. Treatment was of no avail. A possible deduction, drawn from this one case, is that chloromycetin may have a place in the treatment of actinomycosis.

#### REFERENCES

1. Conant, Norman F., and Rosebury, Theodor: *Bacterial and Mycotic Infections of Man*, Edited by Rene J. Dubos, Philadelphia, J. B. Lippincott Co., 1948, p. 579.
2. Eggston, Andrew A., and Wolff, Dorothy: *Histopathology of the Ear, Nose and Throat*, Baltimore, The Williams & Wilkins Co., 1947, p. 911.
3. Zinsser's *Textbook of Bacteriology*, Smith and Martin, Ed. 9, New York, Appleton-Century-Crofts, Inc., 1948, p. 830.

## XXIII

### FIBROSARCOMA OF THE LARYNX

#### REPORT OF A CASE IN A CHILD

DANIEL MILLER, M.D.

BOSTON, MASS.

Neoplasms of the larynx are certainly not uncommon, but fibrosarcoma is only infrequently seen in any large tumor clinic. Serious neoplasms of the larynx in children appear rarely in the otolaryngological literature.<sup>1-46</sup> We are presenting a case of fibrosarcoma of the larynx necessitating laryngectomy occurring in a 7-year-old boy. No account of a younger laryngectomized child was found in the literature.

Cancer has become the second ranking cause of death from disease in children between one and 14 years of age according to the Metropolitan Life Insurance statisticians.<sup>38</sup> Jackson and Jackson<sup>13</sup> reported in their survey in 1941 one death from cancer of the larynx in a boy under four years. Rigby and Holinger<sup>35</sup> in 1943 described a case of fibrosarcoma of the larynx in an infant aged 17 days. The infant died soon after a tracheotomy. The diagnosis of fibrosarcoma was not made until autopsy. Glick,<sup>7</sup> in 1944, reported a case of rhabdomyosarcoma in a 10-year-old boy with an excellent result by removal through laryngofissure. The other early neoplasm, not actually a true malignancy, is the solitary neurofibroma of the larynx reported by Smith<sup>37</sup> in a 6-year-old boy. This was removed by a lateral neck approach and was found to arise from the right aryepiglottic fold.

Fibrosarcoma is a term given to a rather closely related group of tumors, mainly of neurogenic origin. However, there are various types of these tumors which differ in regard to their origin, histology and clinical behavior. It is reputedly a tumor composed of fiber-forming connective tissue cells and is considered highly malignant except when it develops in the skin. Fibrosarcoma of the soft parts is an extremely serious disease with a high mortality rate. Most investigators feel that the deeply placed fibrosarcomas, especially those arising from the muscles and muscle sheaths, are the more

---

Presented before the New England Otolaryngological Society, Boston, Massachusetts, April 27, 1949.

malignant. Stout<sup>43</sup> feels that the periosteal cases are relatively benign and seldom metastasize. In his opinion all of the cases of apparent endosteal fibrosarcomas of bone are either osteogenic sarcomas or chondrosarcomas.

In a study by Taylor and Nathanson,<sup>42</sup> lymph node metastasis was found to be relatively rare, there being only 5-8% in a series of 280 cases of fibrosarcomas in various locations. The metastatic cases showed no regard for the site of the origin of the tumor, or age or sex of the patient. Breast fibrosarcoma seemed least likely to metastasize to regional nodes in their series. Local recurrences are frequently due to invasion of the perineural lymphatics by the sarcoma, as noted by Geschickter.<sup>46</sup> The local recurrent type was found to be most likely to metastasize to the regional nodes. As a result of their studies Taylor and Nathanson<sup>42</sup> have decided that lymph node metastasis seems most likely to occur when the local tumor is large and ulcerated, and especially when it has recurred following primary attempt at surgical removal. They feel that regional node dissection should be decided on only if gross evidence for such metastasis is present.

Stout<sup>43</sup> classified the fibroblastic tumors into three types: (1) the non-neoplastic hyperplasias, such as seen in the true keloids; (2) the fibrous groups, which may or may not be neoplastic, and are almost invariably benign, but which may precede the development of true fibrosarcomas. Examples of these are the true fibromas which are usually intradermal. (3) The third group, the true fibrosarcomas, are outstanding in that these are the infiltrative type. Most of these are well differentiated and are characterized by steady, slow infiltrative growth often lasting many years, or by intermittent growth with long, static periods intervening between times of more rapid proliferation. Occasionally some become less well differentiated and are characterized then by more rapid growth.

A true diagnosis is obtained by biopsy whenever possible since the treatment depends on the type of tumor. The high recurrence rate of 60% for soft part tumors is generally attributed to inadequate local surgery, because some surgeons do not appreciate that fibrosarcoma is a tumor which infiltrates beyond its palpable confines. The optimum time to cure these patients is when they are first encountered. Sacrifice of a little extra tissue on all sides and in the depths at the first operation may prevent subsequent operation and perhaps save a life. If the tumor is anaplastic with many mitoses the case is more urgent and the most radical surgery is indicated, even to an amputation of an extremity or a total laryngectomy. Radiotherapy has proven to be of practically no help in these tumors

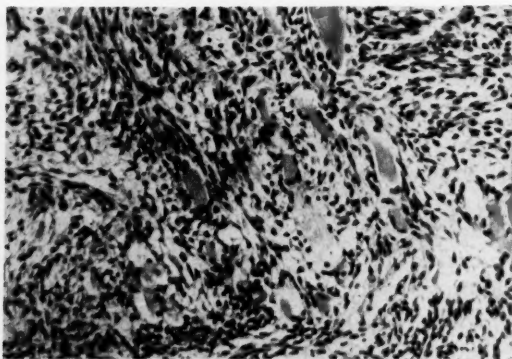


Fig. 1.—Photomicrograph of section of specimen from the larynx.

although an occasional case has responded. Stout<sup>43</sup> has reported two patients who apparently did well, one a 2-year and 8-month-old girl with a well differentiated sarcoma filling the antrum. This child is well eight years and nine months after roentgen therapy. The other was a 28-year-old woman with a well differentiated fibrosarcoma of the trachea 2.5 cm below the vocal cords. She has remained well and symptomless six years after roentgen therapy. Therefore, although most fibrosarcomas do not respond well to radiation therapy, not all of them are completely resistant neoplasms.

#### REPORT OF A CASE

J. L., aged 7, was seen at our office for the first time in June, 1946, with a history of hoarseness since tonsillectomy and adenoidectomy which was done two years previously elsewhere. At first the hoarseness appeared mainly after colds but in recent months had been more or less constant. Because there was a fresh upper respiratory infection present at the time of his first examination, a conservative regime was suggested and he was told to return when the infection had cleared for direct laryngoscopy under general anesthesia. He did not return until September of 1946 at which time his family physician reported that he had developed a wheezing type of breathing which did not respond to drugs commonly used in asthma. He was advised to go into the hospital immediately and a direct laryngoscopy was done.

Under nitrous oxide-oxygen anesthesia, the larynx was exposed with a Jackson laryngoscope, and revealed a large, pale, fleshy, smooth mass, practically filling the laryngeal box. A No. 2 bronchoscope

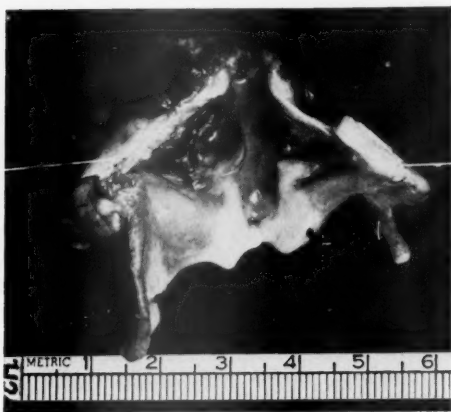


Fig. 2.—Photograph of gross specimen.

was passed because of marked dyspnea during the anesthesia. A tracheotomy was done immediately and a specimen taken for biopsy. The biopsy was reported as fibrosarcoma of the larynx with evidence of mitosis being present (Fig. 1). Because of the extreme youth of the patient and because the actual pedicle of the tumor was not easily observable at the original direct laryngoscopy, it was decided to prepare for total laryngectomy but if possible to do only a laryngofissure in order to save his larynx. There were no glands palpable in the neck at any time.

On September 21, 1946, it was necessary to do a total laryngectomy in the following manner. Under ether anesthesia given through the tracheotomy opening, a midline incision was made and on reflecting the sternothyroid muscles, it was noted that the neoplasm had already extended through the left side of the cricothyroid membrane to the strap muscles. On splitting the larynx, the growth was found to arise from the left half of the larynx extending to the anterior commissure and completely filling the larynx (Fig. 2). Because of the large extent of the tumor the "T" incision was made and a complete laryngectomy was performed by Dr. Philip E. Meltzer. The anterior group of muscles were dissected from their attachments as well. The pharyngotomy opening was closed with interrupted sutures and drains were left in the lateral recesses of the wound.

The child was returned to bed in good condition but that night developed marked difficulty in breathing and cyanosis. We were

called by the house officer who stated that the child had turned blue and was practically unable to breathe. Examination of the chest showed complete lack of aeration in the entire left chest and right lower lobe. The child was placed over the edge of the bed and a bronchoscope was prepared for insertion. On removing the tracheotomy tube a crust was found at its terminal portion completely obstructing the trachea. Inadequate local care of the tube by the nurse had precipitated this emergency. The nurse had been using a very tiny infant's catheter as part of her suction apparatus. Evidently this had been going down through the tube but allowed the crust to form about the terminal portion of the tube thereby giving her a false sense of security that the tube was clear. After a while the crust became large enough to act as a flutter valve and finally closed it off practically entirely.

The lungs immediately re-expanded on withdrawing the tube. Oxygen was given and the child responded very nicely. The bronchoscope was passed anyway but no blood or fluid could be seen in any of the terminal bronchi. However, the trauma of this incident led to another complication in two or three days: a huge fistula in the pharyngotomy opening, evidently as a result of the added tension on the sutures in the pharynx and esophagus during the bronchoscopy. Marked loss of fluid began to take place through the fistula and the laryngectomy dressings frequently became soaked throughout the day. The feedings which were being given through the Levine tube were changed to increase the fat content so as to bring up the caloric intake. Vitamin B complex along with a vitamin A-D combination were also given. Fluids were also increased to make up for the additional loss of the fluids through the fistula but the dehiscence continued to drain. The fistula measured about 4 cm in length.

A modified Thomas collar was fashioned from cardboard and padded with cotton wadding held by gauze. This was fitted snugly around the neck. A small roll of 2-in. bandage was placed against the fistula for added local compression. It was hoped that this fixation and compression of the neck tissues would facilitate the healing by preventing the ooze of pharyngeal secretions from undermining and macerating the tissues. This fortunately proved the case, because within one week the fistula had been reduced from about 4 cm in length to about 1 cm. After another week it had closed completely. The patient was on penicillin therapy all through this period. He was discharged on his forty-third hospital day, 31 days after laryngectomy.



Fig. 3.—J. L., aged 10.

He was immediately started on esophageal speech-training by our specialist in this field, Mrs. Mary Doehler, and within two weeks was able to speak many words. A few weeks later he was back at school and reading to his classmates. He proved to be a very apt pupil for esophageal speech through the efforts of this most capable teacher, herself a laryngectomized patient. He is now able to sing songs with a fair range of pitch and voice quality. This boy is now 10 years of age (Fig. 3) and has continued to develop physically and mentally as any other youngster and has maintained his relations with other children of his age practically as well as previously.

The patient has been followed closely under our supervision and to date there has been no evidence of recurrence.

In Schall's recent report<sup>45</sup> of 500 neoplasms of the larynx at the Massachusetts Eye and Ear Infirmary over a 15-year period, there was no record of any patient with fibrosarcoma or carcinoma in the first or second decade.

#### SUMMARY

A case of fibrosarcoma of the larynx is reported in a boy of seven years of age. Total laryngectomy was performed and two years and nine months following surgery the patient is clinically free of the disease.

20 CHARLESGATE WEST.

## REFERENCES

1. Craig, R. H.: The Treatment of an Early Malignant Condition of the Larynx by Electrodesiccation and Radium, *Arch. Otolaryng.* 12:39-43 (July) 1930.
2. Cutler, M.: Concentration Radiotherapy of Cancer of the Larynx, *J. A. M. A.* 124:967-976 (Apr.) 1944.
3. Figi, F. A.: Sarcoma of the Larynx, *Arch. Otolaryng.* 18:21-22 (July) 1933.
4. Figi, F. A.: Laryngeal Malignancy; Conservative and Radical Treatment, *Radiol. Rev.* 68:183-190 (Sept.) 1936.
5. Figi, F. A., and New, G. B.: Carcinoma of the Larynx in the Young, *Arch. Otolaryng.* 37:425-429 (Mar.) 1943.
6. Garfin, S. W.: Cancer of the Larynx, *New England J. Med.* 213:1109-1123 (Dec.) 1935.
7. Glick, H. N.: An Unusual Neoplasm in the Larynx of a Child (Rhabdomyomyxosarcoma), *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 53: 699-704 (Dec.) 1944.
8. Graham, H. B.: The Pathology of Carcinoma of the Larynx, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 41:453-459 (June) 1932.
9. Graham, H. B.: The Treatment of Carcinoma of the Larynx, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 41:898-902, 1932.
10. Hammond, A. E.: Diagnosis and Treatment of Cancer of the Larynx, *Arch. Otolaryng.* 35:1-19 (Jan.) 1942.
11. Hertz, C. S.: Malignant Disease of the Face, Buccal Cavity, Pharynx and Larynx in the First Three Decades of Life, *Proc. Staff Meet. Mayo Clinic* 15:152-156 (Mar.) 1940.
12. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, *J. A. M. A.* 111:1986-1992 (Nov.) 1938.
13. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, *Arch. Otolaryng.* 33:45-65 (Jan.) 1941.
14. Johnson, W. O.: Intrinsic Carcinoma of the Larynx, *Am. J. Surg.* 11:16-22 (Jan.) 1931.
15. Kernan, J. D.: Two Cases of Early Carcinoma of the Larynx and a Number of Cases of So-called Adenoma of the Bronchi, Apparently Cured by Diathermy, *Laryngoscope* 45:760-768, 1935.
16. Kirch, W.: Carcinoma of the Larynx in the Young, *Proc. Staff Meet. Mayo Clinic* 4:345 (Dec.) 1929.
17. Lewis, F. O.: Cases of Cancer Involving the Larynx, Tonsil, and Ear, *Surg. Clin. N. Amer.* 7:365-368, 1927.
18. Lewis, F. O.: Cancer of the Larynx, *Pennsylvania M. J.* 35:763-768 (Aug.) 1932.
19. Looper, E. A.: Laryngectomy for Cancer of the Larynx, *Surg., Gyn. and Obst.* 64:524-531 (Feb.) 1937.
20. Looper, E. A.: The Surgical Treatment of Cancer of the Larynx, *South. M. J.* 31:367-374 (Apr.) 1938.
21. McCart, H.: Surgical Treatment of Cancer of the Larynx, *Canad. M. A. J.* 55:365-368 (Oct.) 1946.
22. McDougall, C.: Endothelioma of the Larynx, *South M. J.* 19:317-319 (Apr.) 1926.
23. Mackenty, J. E.: Cancer of the Larynx, New York. (Reprint with no other information.)

24. Mackenty, J. E.: Laryngeal Cancer, *Arch. Otolaryng.* 9:237-244 (Mar.) 1929.
25. Mackenty, J. E.: Cancer of the Larynx, *Arch. Otolaryng.* 10:585-602 (Dec.) 1929.
26. Mackenty, J. E.: Malignant Disease of the Larynx, *Arch. Otolaryng.* 20:297-328 (Sept.) 1934.
27. Madden, J. J.: Otolaryngologic Case Reports; Laryngeal Conditions, *Laryngoscope* 46:569-573 (Aug.) 1936.
28. New, G. B.: The Status of Thyrotomy for Carcinoma, *ANNALS OF OTOL-OGY, RHINOLOGY AND LARYNGOLOGY* 40:360, 1931.
29. New, G. B., and Waugh, J. M.: The Curability of Carcinoma of the Larynx, *Surg., Gyn. and Obst.* 58:841-844 (May) 1934.
30. New, G. B., and Hertz, C. S.: Malignant Disease of the Face, Mouth, Pharynx and Larynx in the First Three Decades of Life, *Surg., Gyn. and Obst.* 70:163-169 (Feb.) 1940.
31. Orton, H. B.: Cancer of the Larynx, *Arch. Otolaryng.* 28:153-192 (Aug.) 1938.
32. Orton, H. B.: Diseases of the Larynx, Material Abstracted During the Year 1939, *Laryngoscope* 50:89-163 (Feb.) 1940.
33. Pancoast, H. K.: Some Observations on the Radiation Treatment of Carcinoma of the Larynx, *Am. J. Roentgenol. and Radium Therapy* 12:217-219 (Sept.) 1924.
34. Quick, D.: Carcinoma of the Larynx: Janeway Memorial Lecture, *Am. J. Roentgenol. and Radium Therap.* 38:821-853 (Dec.) 1937.
35. Rigby, R. G., and Holinger, P. H.: Fibrosarcoma of the Larynx in an Infant, *Arch. Otolaryng.* 37:425-429 (Mar.) 1943.
36. Schall, L. A., and Jesberg, N.: Unusual Laryngeal Lesions, *ANNALS OF OTOL-OGY, RHINOLOGY AND LARYNGOLOGY* 56:904-910 (Dec.) 1947.
37. Smith, T. T.: Solitary Neurofibroma of the Larynx, *Arch. Otolaryng.* 39:144-151, 1944.
38. *Statist. Bull. Metr. Life Insur. Co.* 30:5-8, 1945.
39. Tucker, G.: Early Intrinsic Cancer of the Larynx, Diagnosis and Treat-ment; Observations on Laryngofissure as a Method of Treatment in a Series of Cases, *ANNALS OF OTOL-OGY, RHINOLOGY AND LARYNGOLOGY* 41:36-51 (Mar.) 1932.
40. Tucker, G.: Cancer of the Larynx, *Surg., Gyn. and Obst.* 46:303-308, 1938.
41. Van Loon, E. L., and Diamond, S.: Neurofibroma of the Larynx, *ANNALS OF OTOL-OGY, RHINOLOGY AND LARYNGOLOGY* 51:122-126 (Mar.) 1942.
42. Taylor, G. W., and Nathanson, I. T.: *Lymph Node Metastasis*, New York, Oxford University Press, 1942.
43. Stout, Arthur Purdy: Fibrosarcoma—the Malignant Tumor of Fibroblasts, *Cancer* 1:30-63 (May) 1948.
44. Nathanson, I. T., and Welch, C. E.: Life Expectancy and Incidence of Malignant Disease, *Am. J. Cancer* 31:598-608, 1937.
45. Schall, L. A., and Ayash, J. J.: Cancer of the Larynx, *ANNALS OF OTOL-OGY, RHINOLOGY AND LARYNGOLOGY* 57:377 (June) 1948.
46. Geschickter, C. F.: Tumors of Peripheral Nerves, *Am. J. Cancer* 25:377, 1935.

## XXIV

### AN UNUSUAL COURSE OF AN OPEN SAFETY PIN IN A BABY 8 MONTHS OLD

PETER D. LAELLA, M.D.

NEW ROCHELLE, N. Y.

AND

DAVID H. JONES, M.D.

NEW YORK, N. Y.

We are presenting this case report because of the unusual course of events of an open safety pin swallowed by an eight-month-old infant. Vinson<sup>7</sup> states that foreign bodies are seldom encountered in the esophagi of infants less than nine months of age.

Our case report is that of an eight-month-old infant in which fluoroscopy revealed a safety pin at the level of the lower cervical vertebrae. X-ray films were taken and revealed an open safety pin which measured 2.8 cm on the radiograph with the open end cephalad, located in the esophagus about 1.5 cm below the cricopharyngeus.

Five hours later under drop ether with the child in the Jackson position, a Cameron anterior commissure speculum was introduced. The cricopharyngeus was easily passed. No safety pin was visualized. The esophagus was then explored to the cardia but no safety pin was seen. Fluoroscopy showed the pin in the stomach. No further manipulation was thought feasible at this time, and the child was returned to its room. Daily x-ray films showed the pin moving about in the stomach but not presenting at the pylorus. The child showed no signs of distress. There was no vomiting and no crying and it took its feeding without difficulty.

At the end of seven days it was thought advisable to send the baby home and have the pediatrician follow the child's progress by fluoroscopy since the pin was freely movable in the stomach and since the infant was showing no signs of distress. The pin was non-magnetic; we did not have a biplane fluoroscope so that gastroscopic

---

Read before the otolaryngological section of the New York Academy of Medicine, January 18, 1950.

removal could not be done and the parents refused to move the child to another hospital. We did not feel that a gastrostomy should be done at this time. The pediatrician kept us informed of the child's progress. It never showed any symptoms through a period of 41 days. It ate well, slept well, and had normal bowel movements (no cathartics were used). There was no vomiting and fluoroscopy showed that the pin was moving about in the stomach but not presenting at the pylorus. On the forty-first day it was decided that a gastrostomy be done and the child was readmitted to the hospital. No x-ray films had been taken and no fluoroscopy done for about ten days. On the morning of the forty-second day an x-ray film showed the pin in the lower end of the esophagus. The open end was directed caudad approximately 3.5 cm above the cardiac orifice.

Without anesthesia, the baby in Jackson's position (that is, the Trendelenburg position with the operator and assistant sitting on the floor) a Cameron anterior commissure speculum was passed and the pin identified by its presenting spring. It was grasped with a Koehler forceps and removed. There was no trauma. The child was discharged within 48 hours, fully recovered.

This case presented to us a number of interesting problems:

1. No endoscopic procedure should be carried out for the removal of a foreign body unless x-ray films have been taken within one-half hour. We have always been fully aware of this but must admit that at this time we were guilty of omission. X-ray films were taken at two o'clock and we attempted removal of the pin at about seven.

2. An eight-month-old infant moving, struggling, and crying presents a rather "fast-moving target." The size of the infant necessitates the use of small lumen 'scopes, and the foreign body was a sharp, pointed one, which could very easily perforate the esophagus of the infant. These facts prompted the use of general anesthesia in the first attempt at removal. We will never know if the safety pin was dislodged from the esophagus into the stomach spontaneously between the hours of two and seven, or whether it slipped down while the baby was completely relaxed under general anesthesia.

3. Our third problem, the most difficult, was (a) would the pin after reaching the stomach pass the rest of the way safely and successfully? (b) How long should we wait for it to present itself at the pylorus? (c) When should a gastrostomy be done?

One of us (D. H. J.) felt that as long as the safety pin was freely movable in the stomach and the child showed no signs of

distress, a period of watchful waiting was indicated. A number of general surgeons, physicians, pediatricians, and otolaryngologists expressed varied opinions. Some recommended waiting three to four days, others two to three weeks, still others, periods in between. It was interesting to note that the large majority of general surgeons suggested four to six days, whereas the pediatricians were by a large majority in favor of waiting three to four weeks.

In searching the literature we find a variety of experiences. Vinson<sup>3</sup> reports a case of an open safety pin passing spontaneously in thirty-six hours. Myers<sup>5</sup> stresses the danger of removal of open safety pins by gastrostomy in infants and advocates gastroscopic removal or a period of watchful waiting. Benedict<sup>6</sup> reports early regurgitation of open safety pins. Caswell<sup>10</sup> reports two open safety pins that passed spontaneously in 48 hours. Jackson and Spencer<sup>9</sup> in 1921 reported a case of a six-month-old infant with two closed safety pins 2 in. long in the stomach for 27 days which would not pass and had to be removed by gastroscopy. Of all the reports only one was found that approximated our present case. This is the one reported by Jackson and Spencer in 1921. Their case was that of a twelve-month-old baby with an open safety pin in the stomach for a period of seven weeks, which was then regurgitated into the esophagus point down and was removed by means of the esophagoscope. Jackson at this time stated that the regurgitation of an open safety pin into the esophagus is a rarity. He further stated that in the absence of symptoms one can wait up to two months before gastrostomy. He concluded that a foreign body that reaches the stomach will pass and that removal from the stomach is indicated in but two types of cases: (1) Those in which the foreign body is large in size in relation to the patient; (2) those in which a watchful waiting period of up to eight weeks has demonstrated that the foreign body will not pass.

With all these different opinions on hand it seems that the choice of the length of a waiting period rests with the surgeon faced with an individual problem, under given working conditions. Our choice of a prolonged period was based on our own individual problem and is not meant as a criterion for other similar cases.

524 NORTH AVENUE.

#### REFERENCES

1. Jackson, Chevalier L.: Foreign Bodies in Air and Food Passages, *Postgrad. Med.* 4:281 (Oct.) 1948.
2. Paterson, Ritchie D.: Open Safety Pin in Infant's Gullet, *Brit. M. J.* 1:222 (Feb. 17) 1945.

3. Vinson, Porter P.: Spontaneous Passage of Open Safety Pin from Esophagus into Stomach with Point of Pin Downward, *Virginia M. Month.* 73:329-330 (July) 1946.
4. Bunker, Paul G.: Method of Rotating the Open Safety Pin in the Esophagus, *Arch. Otolaryng.* 37:78-81 (Jan.) 1943.
5. Myers, E. Lee: Open Safety Pin in Stomach; Removal by Endoscopic Measures Assisted by Single Plane Fluoroscopy, *Laryngoscope* 51:299-303 (Mar.) 1941.
6. Benedict, Edward B.: Vomiting of an Open Safety Pin, *New England J. Med.* 203:484 (Apr. 20) 1944.
7. Vinson, Porter P.: Foreign Body—Open Safety Pin in Esophagus of Infant, *Virginia M. Month.* 75:363 (July) 1948.
8. Norris, Charles M.: Foreign Bodies in the Air and Food Passages. A Series of 250 Cases, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:1049 (Dec.) 1948.
9. Jackson, C., and Spencer, W. W.: Open Safety Pin in Esophagus of Infant, *J. A. M. A.* 76:577-579 (Feb. 26) 1921.
10. Caswell, H. O., and Magerns, P. J.: Open Safety Pin in Stomach and Intestines, *Wisconsin M. J.* 20:83 (July) 1921.

## FIBROMA OF THE NASOPHARYNX

## A CASE REPORT

KENNETH L. DIEHL, M.D.

ROCHESTER, N. Y.

Fibroma of the nasopharynx, in itself, is a rare type of tumor. Very few rhinologists in their lifetime see more than a few cases. Cancer of the nasopharynx represents about 2% of all malignant growths of the head and neck.<sup>1</sup> Nasopharyngeal fibroma, while not actually classified as being malignant, makes up a much smaller percentage.

As expressed by Eggston and Wolff,<sup>2</sup> the most commonly accepted theory concerning their etiology, is that these tumors are due to an unequal development of the cranial bones during fetal life. When the patient reaches puberty, with the stimulation of bone growth, the congenital defect causes hypertrophy instead of normal bone formation. Other writers, especially Furstenberg,<sup>3</sup> feel that many nasopharyngeal fibromas are diagnosed as such when they are actually fibrosed polypi or angiomas. At any rate, the majority of these nasopharyngeal fibromas tend to regress as adulthood is attained, if death does not occur from hemorrhage or extension. Som and Neffson<sup>4</sup> differentiate the fibroma into a juvenile type and a cellular type. The juvenile type is very vascular and contains a predominance of immature fibroblasts and at the same time lacks a great number of fibrous strands. Lack of muscle fibers in the walls of the large vessels, enclosed in dense fibrous tissue, explains the tendency to hemorrhage. It is because of this tendency to bleed that any suspicion of a juvenile nasopharyngeal fibroma would lead one not to perform a biopsy. The cellular type, on the other hand, seems to be a more mature tumor, with mature fibroblasts, and a distinct fibrotic capsule, with less vascularity. This type is apparently that described by Figi<sup>5</sup> as a periosteal fibroma. This type has no predilection insofar as age and sex are concerned as does the juvenile fibroma. The clinical and pathological differentiation between the juvenile and the cellular is slight, so that it is extremely difficult to make any hard and fast rule of difference.

Figi,<sup>5</sup> in 1940, reported one of the largest series of nasopharyngeal fibroma cases in the literature. Fifty-eight were in men and only

five were in women. Also, of this number, only two were cellular fibromas; these were intranasal, extending into the nasopharynx. Shaheen,<sup>6</sup> in 1930, reported nasopharyngeal fibromas in men 48, 60, and 70 years of age. According to Figi,<sup>5</sup> however, there is some doubt as to the basic pathology of his series.

The symptoms are usually nasal obstruction of one or more nostrils, and bleeding which may be from either the nose or the pharynx. The mass usually grows to fill the nasopharynx, then extends into the posterior nares, causing pressure on, and growing into, the ethmoid labyrinth, the maxillary sinus and the sphenoid sinus. Only later is it seen to extend down into the pharynx below the soft palate or causing the soft palate to bulge. A complication which frequently occurs is obstruction of one or both eustachian orifices, resulting in deafness, pain in the ears, and otorrhea. Finally, as a result of growth through the foramen lacerum, the cribiform plate, or the superior orbital fissure, there may be pressure necrosis, bone erosion and death.

The juvenile nasopharyngeal fibroma is of a deep purplish or grayish color. It is usually rounded, of a firm cartilaginous consistency and may be slightly nodular. It is usually attached by a broad base in Rosenmüller's fossa, high on the posterior wall of the nasopharynx, or it may arise from the periosteum covering the basilar process of the occipital bone or the body of the sphenoid. More infrequently it arises from the upper two cervical vertebrae, the internal pterygoid plate, the region of the foramen lacerum, or the pterygomaxillary fossa. The cellular or periosteal type is quite similar to the juvenile type in its physical characteristics except that it may be slightly more firm, a little lighter in color, because of its lesser vascularity, and sometimes has a pedicle, although these characteristics cannot be judged as diagnostic.

#### REPORT OF A CASE

Mrs. E. F., a white female aged 52, was seen in my office complaining only of serosanguineous drainage from the left ear for one day. On questioning, further symptoms were elicited. These were, nasal obstruction and discharge for about six to eight months, with slight difficulty in hearing and fullness in the left ear for the same length of time. She also stated that for the past month she had had some trouble swallowing solid foods. For almost two years the family had noticed a slowly increasing nasal twang in her speech.

Examination revealed some blood and serous material in the left ear canal; the drum was of a bluish cast, making it appear as if the middle ear contained blood. The right ear was within normal limits.

The nasal cavity contained a great deal of thin mucus and both the middle and inferior turbinates had a pale, boggy appearance. The sinuses were clear on transillumination. Examination of the pharynx revealed a large tumor originating in the nasopharynx. The soft palate was pushed forward, and the tumor mass could be seen extending about 2 cm below it. The larynx and hypopharynx were normal. The mass was quite firm, and no definite pedicle could be palpated, although it was thought to arise from the left nasopharyngeal wall. It was grayish in appearance, contained a few nodular elevations over its surface and seemed to be covered with normal epithelium. The consulting roentgenologist decided that x-ray or radium therapy was not indicated. It was therefore decided that surgical excision should be attempted.

Necessary pre-operative precautions were taken to minimize bleeding and to prepare for it. That is, large doses of vitamin K were given, the patient was typed and cross-typed, blood was obtained and ready, and a set-up prepared for ligation of the external carotid arteries. The size of the tumor necessitated splitting the soft palate. The base was a wide, short pedicle originating posterior to the left eustachian orifice and filling Rosenmüller's fossa. The base was severed as close to the left nasopharyngeal wall as possible by means of electrocautery, with surprisingly little bleeding. Following the removal, examination of the site did not reveal any remaining tumor mass on the left nasopharyngeal wall. There was no extension to the bony wall, the fibroma apparently coming from the subepithelial layer. The site of removal at the lateral wall was at the level of the torus tubarius, partially involving it and obliterating the eustachian orifice. One persistent bleeding vessel was ligated, the mucous membrane edges were approximated, the soft palate was sutured, and the patient left the operating room in good condition. The operation was performed under endotracheal oxygen and ether; the anesthetic was discontinued while the electrocautery was being used. The postoperative course was uneventful. The hearing in the left ear returned to normal, and six months after operation, no residual effects or recurrence of tumor was noted.

Following is the pathologist's report:

*Pathologic Examination—Gross:* The specimen consists of a firm, ovoid mass, measuring 6 x 5 x 4 cm and weighing 44.5 gm. It is covered by hemorrhagic mucous membrane, and the cut surface reveals a dense white tissue arranged in whorls.

*Microscopic:* The microscopic examination shows interweaving bundles of fibrous tissue in which there are numerous small blood

vessels. Some fat vacuoles are present and the surface is covered by columnar respiratory type of epithelium which shows focal metaplasia to a transitional type.

*Diagnosis:* Fibroma, nasopharynx.

Because the microscopic section revealed a predominance of fibrous tissue, with too few immature fibroblasts, the pathologist felt that he could not definitely diagnose this as a juvenile type. At the same time the lack of a great number of mature cells, with the presence of numerous small blood vessels prevented him from definitely diagnosing it as of the cellular type. Since complete excision was accomplished, and the origin of the tumor mass seemed to arise from the soft tissue of the lateral nasopharynx, one might be led to believe that this was a fibrosed angioma or polyp.<sup>3</sup>

#### SUMMARY

A case report of a nasopharyngeal fibroma is presented because of several unusual features:

1. A fibromatous tumor of extremely large size.
2. A very unusual age for this type of tumor, 52.
3. Occurrence in a woman.

277 ALEXANDER STREET.

#### REFERENCES

1. Martin, H. E., and Blady, J. V.: Cancer of Nasopharynx, *Arch Otolaryng.* 32:692 (Oct.) 1940.
2. Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose, and Throat, Baltimore, The Williams and Wilkins Company, 1947, p. 749.
3. Furstenberg, A. C.: Malignant Neoplasms of the Nasopharynx, *Surg. Gyn. and Obst.* 66:400, 1938.
4. Som, Max L., and Neffson, A. H.: Fibromas of the Nasopharynx: Juvenile and Cellular Types, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 49:211, 1940.
5. Figi, F. A.: Fibroma of the Nasopharynx, *J. A. M. A.* 115:665-671 (Sept.) 1940.
6. Shaheen, H. B.: Nasopharyngeal Fibroma, *J. Laryng. and Otol.* 45:259-264 (Apr.) 1938.

# New Instrument

---

XXVI

## A NEW INSTRUMENT FOR PERFORMING TONSILLECTOMY AND OTHER OTORHINOLARYNGOLOGICAL OPERATIONS

SHINJI YOSHIDA, M.D.

FUKUOKA, KYUSHU, JAPAN

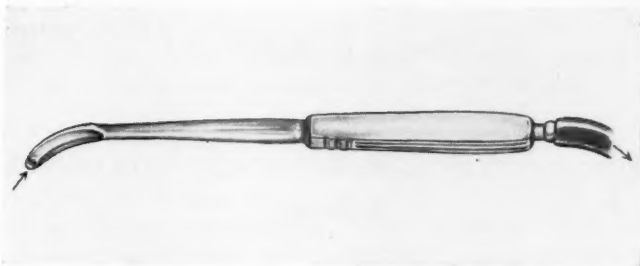
About 20 years ago the author was impressed by the difficulties which were sometimes encountered in the performance of tonsillectomy. A few of those difficulties were excessive bleeding, aspiration of blood with consequent coughing and gagging and the other pulmonary complications incidental to tonsillar surgery. As practically all tonsillectomies in Japan are done under local anesthesia, this presented a problem. After 11 years of experimentation, I have finally devised the instrument shown in the figure.

This instrument consists of a dissector 20 cm in length with a spatulous dissecting end with serrated edges and a hollow body. A small orifice in the center of the tip (arrow) provides for constant suction during operation. The suction tube is attached to the handle end of the dissector.

In the performance of a tonsillectomy, the anterior pillars of both tonsillar fossae are incised in the usual manner and the tonsil grasped with a locking-type, long curved tonsil forceps. The tonsil is then lightly put on a stretch and the dissector with suction connected to it is used to gently dissect the tonsil from its bed. This is practically a bloodless procedure due to the fact that suction is constantly applied to the operating field.

Not only can this dissector be used for tonsillectomy but also for operations in the ear, nose and throat. Possibly a somewhat different version of this instrument could be applied to the other specialties of surgery.

DAIMYO MACHI 3-223.



Edited by Lt. Colonel S. W. French, III, MC, 118th Station Hospital, APO 24-5, c/o Postmaster, San Francisco, California.

Editor's Note: I have seen Dr. Yoshida use this instrument many times and the almost absolute hemostasis which is constantly present, the lack of coughing and gagging by the patient, and the ease with which tonsils are removed with this dissector are truly something to behold. It is believed that this instrument may find a valuable place for itself in the armamentarium of surgical instruments used by otorhinolaryngologists throughout the rest of the world.

# Society Proceedings

---

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

*Meeting of Monday, March 7, 1949*

THE PRESIDENT, DR. WILLIAM A. SMILEY, IN THE CHAIR

### Electroencephalography in Diagnosis of Hearing Loss in the Very Young Child

RICHARD E. MARCUS, M.D. (By Invitation)

E. L. GIBBS, M.D. (By Invitation)

AND

F. A. GIBBS, M.D. (By Invitation)

(Abstract)

Hearing in the young child may become seriously, impaired or lost completely as the result of numerous local or systemic diseases and certain poorly understood hereditary or intra-uterine influences. Because of the tremendous social handicap imposed on the child with defective hearing, an early, definitive diagnosis is imperative so that specific therapy and proper educational techniques may be employed.

This study is concerned with the nonspecific *arousal response* noted on the electroencephalographic tracing when loud sound is used as a stimulus while the child or infant is asleep. If the sound is perceived, a definite change in the brain wave pattern becomes evident. Stimulation of olfactory and taste sensation was used as a control in 19 patients whose ages ranged from 17 months to 9 years. Thirteen of these patients had no previous demonstrable hearing and six had reduced hearing, but defective speech. Four of the 13 considered to have no hearing showed an electroencephalographic arousal response to loud sounds. An incidental but potentially important finding was that of intracranial damage as shown on the electroencephalographic tracing. The finding of intracranial damage may

indicate that some of these children do not have true "peripheral" hearing loss but may actually have one or more types of auditory aphasia. Further studies for calibration of the sound stimulus and correlation of intracranial damage with hearing loss are contemplated.

#### DISCUSSION

DR. THOMAS C. GALLOWAY: Were any of these children tested with conditioned reflexes? This method, developed by Dr. C. A. Aldrich, seems of value in infants. A bell is rung followed by a gentle pinprick. After a few trials the hearing child cries as soon as the noise is made.

DR. SHERMAN SHAPIRO: Might not the depth of slumber and the sedation used influence the degree to which the patients responded?

DR. R. D. RUSSELL: I have been under the impression that substances like acetone and ammonia gave fifth nerve responses. I would like to know how Dr. Marcus separates that from olfactory response.

DR. RICHARD E. MARCUS (closing): In reply to Dr. Galloway, we did not do any such testing. The children were so profoundly deafened that it would seem any conditioning one would attempt would have to be prolonged, using pins or other methods which parents would object to; whereas we can step right into this. The child may put up a fight while the electrodes are placed, but falls into a nap. It is an innocuous procedure. I do not know what more we could learn by conditioning technique in the profoundly deafened child, except that it might be worth while for comparison.

In reply to Dr. Shapiro, the relationship of response to the depth of slumber and sedation has bothered us. Doctors Gibbs have done some 20,000 electroencephalographic tracings, a fair percentage of which have been under barbiturate-induced sleep. They feel that the effect of barbiturates can be evaluated separately, unless sedation has been carried to the point of anesthesia, in which instance the arousal response to sound cannot be obtained. The depth of sleep, however, is important, and we try to keep the children at the level of light sleep. The depth of sleep can be judged by the type of electroencephalographic tracing. Some of the children fall into deep sleep from which they can be aroused only with greatest difficulty; it is sometimes necessary to shake them to get them awake. When this occurred the absence of the arousal response to sound was discounted; another record was accomplished under more favorable cir-

cumstances, or else the child was not used for the purposes of this study.

Dr. Russell asked about the use of acetone. Acetone may stimulate sensory systems other than olfaction alone, but it did not concern us directly since we were looking for a method of stimulation other than auditory. If the child responded to olfactory or tactile stimulation and not to auditory stimulation, we concluded that he could not hear.

### **Evaluation of Irradiation of Pharyngeal and Nasopharyngeal Lymphoid Tissue**

FRANCIS L. LEDERER, M.D.

(This paper appears in full on page 102 of this issue.)

#### **DISCUSSION**

DR. JOHN R. LINDSAY: This subject came up for discussion last spring at the A. M. A. meeting. In my discussion of Dr. Farrior's paper I pointed out that in our experience of examining the eustachian tube histologically, lymphoid tissue in the eustachian tube proper in anything more than microscopic nodules is rare. On the other hand, the tissue in Rosenmüller's fossa extends up along the tube to a degree greater than is usually realized, 1 cm or more beyond the level of the torus. If one expects an effect from beta rays I am quite sure it would not reach that distance. On the other hand, lymphoid tissue extending into the mouth of the tube does not occur beyond 1 or 2 mm. I was interested to hear Dr. Guild confirm this observation.

If we are trying to get at a pathological condition in the eustachian tube beyond the very mouth of the tube, we are probably dealing with something other than hyperplastic lymphoid tissue. The thesis of the essayists at the A. M. A. meeting was to recommend x-ray therapy in such cases. If one uses x-ray therapy for such a condition in the tube one is not treating hyperplastic lymphoid tissue, but probably the result of former inflammatory reactions.

Did Dr. Lederer wish to infer that the existence of cartilage and bone in the tonsil demonstrated was the result of irradiation? Cartilage and bone have long been known to occur relatively frequently in tonsils and there would be no reason to attribute its presence in this case to irradiation.

DR. G. H. MUNDT: No one is competent to say that he does or does not get satisfactory results. I would differ with Dr. Lederer on one point; if a radium applicator is to be used, I think the otolaryngologist should apply it.

Some of us have used radium as honestly as we know how. We have made repeated audiograms on children and watched them carefully, with special attention to the pharynx. Long before radium was advocated I frequently removed large masses of lymphoid tissue from the nasopharynx in children who had had adequate tonsil and adenoid operations. In some, the improvement in hearing and general condition was most satisfactory. My experience with x-ray in the treatment of tonsils and adenoids has not been satisfactory.

Dr. Lederer spoke of inadequate operations. The faucial tonsils can be taken out satisfactorily, but no one can be certain how much recurrence there will be following surgery of the lingual and pharyngeal tonsils and, by the way, we should eliminate the word adenectomy. Some children receive marked benefit, some get little if any improvement, just as in any method of treatment.

DR. THOMAS C. GALLOWAY: In our office we have used radium rather often in hearing impairment referred from a school survey. We came to it reluctantly, but the results have been about 80% favorable. There were some side effects that were especially good, including help in allergy, as Crowe has pointed out.

The record of x-ray therapy in malignancy is impressive. There are a number of explanations of its action, and there is doubt as to whether it acts on the protoplasm, the nucleus or the blood supply. In infection, there is doubt as to what irradiation actually does. It probably acts to destroy infiltrating white cells and free enzymes, and I am sure it is of considerable value. I have reported 100 cases of infection, carefully studied, in which irradiation was used with much benefit.

I agree with Dr. Mundt that radium can be a carefully applied modality, and in the hands of the trained laryngologist it is possible to apply it accurately; if properly applied it does not disseminate infection. I do not think it can be lightly dismissed or condemned.

DR. FRANCIS L. LEDERER (closing): I had always assumed that cartilage and bone could be found in any tonsillar material that was sectioned, not specifically identified with irradiation changes. In this particular tonsil, radon was actually placed in the tonsil; whether that had any relation I do not know. Dr. Tamari did not identify cartilage and bone as a normal observation in tonsils, and believed it to be the result of irradiation per se. I believe that cartilage and bone

is a normal finding in the irradiated structure of the tonsil. The technique should be adequate for depth. This was a quotation from Dr. Wachowski, and otherwise I am in agreement with what Dr. Lindsay has said. I had hoped that the audience would help me with my indecision, and thought I would hear some testimonials on the therapy. I have no more proof than Schulz and Robbins, and am not willing to say that this is a dangerous treatment in proper hands. However, I contend that otorhinologists have no business in employing radium unless they are qualified in its use, and I do not think the average man knows enough about it. Some men make no difference between radium as an element and radon. I have heard about wonderful results publicized by others, but I cannot brag about my own.

#### **Blood Pressure Changes in Fenestration Surgery**

M. J. TAMARI, M.D.

AND

M. H. CUTLER, M.D.

(This paper appears in full on page 179 of this issue.)

#### **DISCUSSION**

DR. SHERMAN SHAPIRO: This excellent work confirms what has been well known experimentally, and I think it deserves more than passing interest. I would suggest for the sake of completeness that a control series be run on a group of plastic cases under local anesthesia, to show the difference of procedure; the same type of medication might be of value.

DR. ALFRED LEWY: Is there any comparable list of other surgical operations performed under local anesthesia to serve as controls; is this more or less a shock phenomenon?

DR. MAX H. CUTLER (closing): Someone asked how much influence the pre-operative medication had. This was started eight hours before and was rather light, using nembutal and morphine, and we had the feeling that while it did tend to lower the blood pressure it was not sufficient to cause this particular change.

We have performed endaural radical mastoid operations under local anesthesia and in no instance has there been a strikingly low blood pressure. However, in one case, during operation, the anes-

thetist called attention to the sudden drop in blood pressure. The following day that patient had definite nystagmus and symptoms of labyrinthitis, entirely due to surgery.

We hope to continue the study.

*Meeting of Monday, April 4, 1949*

THE PRESIDENT, DR. WILLIAM A. SMILEY, IN THE CHAIR

**The Development of the Otic Capsule in the Region of Surgical  
Fenestration\***

BARRY J. ANSON, PH.D.

(This paper appeared in full on page 739 of the September, 1949, issue.)

DISCUSSION

DR. GEORGE SHAMBAUGH, JR.: Dr. Anson has continued his original and monumental studies of the development of the labyrinthine capsule with this lucid, detailed and beautifully complete account of the horizontal semicircular canal. The clinical application of such fundamental research is not always evident, but in the case of the labyrinthine capsule there are important clinical implications.

As Dr. Anson has so clearly shown, the middle or enchondral layer of the labyrinthine capsule is unique in the rapidity of its formation from fetal cartilage during the fifth month of fetal life, reaching adult dimensions long before birth, and retaining its histologic form without further alteration throughout the life of the individual. It is this inert quality that helps to make possible the creation of a permanently open fenestra in a high percentage of operations. In a recent study of 300 consecutive fenestration operations followed for more than two years after operation, loss of the hearing improvement presumably due to osseous closure occurred in less than 1%. The surgical technique that has yielded this result includes the following features:

---

\*A study carried out in collaboration with Dr. Theodore H. Bast, Department of Anatomy, University of Wisconsin.

1. Enchondralization of the fenestra, by which we mean the wide exposure of the relatively inert enchondral layer by removal of the more active periosteal layer far beyond the margins of the completed fenestra, which is left on top of a dome-shaped elevation. The enchondral bone is easily recognized through the operating microscope by its yellowish color and the coarsely granular appearance imparted by the islands of ossified cartilage. The enchondral bone has been likened to pineapple ice cream as compared to the smoother white color of the periosteal layer resembling vanilla ice cream.

2. The micro-immaculate removal of every particle of bone dust, with the help of continuous irrigation and the operating microscope.

3. Endosteum-hemming of the fenestra, by which we mean the careful preservation of the intact endosteal membrane up to the sharp knife-edge of the fenestra so that it will unite with the periosteum of the skin flap. Thus there is left no naked raw unprotected bone edge from which osteogenesis will occur. The use of a knife to outline a bony lid, as recently described by Lempert, may have the disadvantage of stripping the endosteum and leaving an edge of unprotected bone.

4. The fenestra should be epidermis-periosteum gloved, meaning that as thin a skin flap as possible should hug the bony margins of the fenestra as tightly as possible. The enchondralization technique, leaving the fenestra on top of a dome, ensures this glove-like fit.

5. Burnishing the bone around the fenestra with a smooth hard gold bur seems to help to inhibit osteogenesis, possibly by sealing the openings of the bone capillaries.

6. The prevention of fibrosis by a meticulous aseptic technique, combined with perfect hemostasis, is important, for fibrosis in the perilymph space is frequently followed by osseous invasion of the fibrous tissue.

I should like to inquire about one point not mentioned by Dr. Anson. Of the 2,100 consecutive fenestration operations performed at Northwestern University Medical School, an otosclerotic focus involving the horizontal semicircular canal was observed at operation in 62, an incidence of about 3%. The otosclerotic focus in the enchondral layer is easily recognized under the operating microscope by its whiter, softer, more vascular appearance, as contrasted with the hard yellowish enchondral bone with its islands of ossified cartilage. In some cases the otosclerotic area in the semicircular canal is continuous with the area around the anterior end of the oval

window, but in others the focus in the bony horizontal semicircular canal is discrete, with no connection with the focus at the site of predilection. I should like to ask Dr. Anson if he or Dr. Bast have encountered any areas of unossified cartilage in the semicircular canal region that might act as a nucleus for the development of such otosclerotic changes.

DR. BARRY J. ANSON: We are completing a survey of the otological series in the collection at Northwestern University Medical School in order to determine how frequently such cartilaginous areas persist and where they are commonly located. It is known, of course, that they are normally present in the otic capsule of the fetus at constant sites, and that they are usually replaced by bone in the course of postnatal development. We have found no relationship between such areas and the otosclerotic foci, except in the fissular region.

*Meeting of Monday, November 7, 1949*

THE PRESIDENT, DR. OLIVER E. VAN ALYEA, IN THE CHAIR

**Physiology of Respiratory Resistance**

JOHN S. GRAY, M.D. (By Invitation)

(This paper appears in full on page 72 of this issue.)

**Favorable Results in Bulbar Poliomyelitis Treated as a Problem in  
Respiratory Obstruction**

THOMAS C. GALLOWAY, M.D.

AND

MARTIN H. SEIFERT, M.D. (By Invitation)

(Abstract based on paper published in the J. A. M. A. 141:1, Sept. 3, 1949)

In bulbar poliomyelitis it has been usually assumed that the grave effects were due to direct action of the virus itself. We have quite a different concept of it. Treatment under nearly ideal conditions based on that concept resulted in 15 consecutive recoveries from bulbar poliomyelitis—all the bulbar cases seen among 127 total

cases in nearly three years. This contrasts with a mortality in the same area for the same period, of 24% in bulbar poliomyelitis and 3.4% for all cases. (Our recent results, however, have not been so good in patients hospitalized late.)

Anoxia, with associated carbon dioxide excess, is frequently overlooked as an important cause of symptoms and serious pathologic changes. Especially is this true in secretional obstruction. We believe that the chief difficulty in severe bulbar poliomyelitis is usually interference with the swallowing mechanism, resulting in accumulation of secretion, food, fluid, or vomitus in the airway. This results in anoxia with its central and peripheral effects and in secondary lung changes. These are usually promptly relieved on clearing the airway, if done early.

In the 15 recoveries reported, the following measures have been used:

1. Adequate postural drainage at 25° to 35°.
2. Continuous water suction.
3. Oxygen and parenteral fluids and feeding.
4. Use of the respirator when respiratory failure threatens.
5. Tracheotomy when indicated to prevent dangerous anoxia and CO<sub>2</sub> accumulation, to prevent drowning in secretions, to permit by-passing fluid accumulation, allow aspiration of the lower air-passages, and to make safe the use of the respirator. Tracheotomy was necessary in six of the 15 cases.

The methods for the safeguarding of the airway must be effectively applied before there occurs severe brain damage or irreversible changes in the lung field such as atelectasis, atypical pneumonia and pulmonary edema, which so often kill these patients. We believe that administration of CO<sub>2</sub> is not indicated in respiratory obstruction.

#### DISCUSSION

DR. MARTIN H. SEIFERT: I have been seeing cases of poliomyelitis for about 19 years, and for the past seven years have had charge of the service at Evanston Hospital. We have had about 900 cases, a large proportion of which have been in the last six or seven years. I was fortunate to have in the hospital a man with the courage and decisiveness of Dr. Galloway.

This year, as Dr. Galloway told you, we have not had 100% recoveries, but there are some ameliorating circumstances. We have had 26 cases of bulbar poliomyelitis this year—about as vicious as anything I have seen. We lost five. One patient came in about eight hours before death in a state of cyanotic flush and elevated

blood pressure. Tracheotomy was performed shortly after admission and in spite of everything she developed several asphyctic crises and we lost her. I think in general when you see these spells of cyanosis or anoxia you can just about halve the expectancy of that patient's chance for recovery. In the second case we were not permitted to do tracheotomy early. The third patient came in about the fifth day of the disease, had already had several crises, had cyanotic pallor, and was in shock; tracheotomy was done and about 150 cc of fluid gushed from the tracheotomy wound. He died in three hours. In the fourth patient tracheotomy had apparently been done in time, but he died within a few hours. I cannot help but feel that this was a patient who had enough virus infection to knock him out. The fifth case was that of a pregnant woman who had been in bed because of hemorrhage. She had to have an emergency cesarean section following premature separation of a placenta previa. The next day she went into a crisis and we put her in a respirator, but finally had to perform a tracheotomy and she did not survive.

Actually, the plea I would make is, first of all, for a change in attitude. I survived the first 12 years or so of my contact with poliomyelitis with an attitude that I am sure was not healthy; that bulbar poliomyelitis was a vicious fulminating brainstem infection and was fatal. That is a defeatist attitude. There is something that can be done for it. As Dr. Galloway said, when you see paradoxical breathing it is amazing; this patient had marked paradoxical breathing and when Dr. Galloway put a tube into the trachea that patient breathed normally. We should change our attitude to one of hope and activity. We must include in the polio team a laryngologist with courage and understanding, and the treatment of polio requires teamwork. The use of postural drainage and suction and oxygen is necessary. I would plead for the use of the respirator with bulbar polio, but unless one can keep a clear airway it is dangerous. Wangenstein suction is of utmost importance to keep fluid and food out of the mouth; patients may not be able to handle it if they vomit. After five or six days, if they are beginning to swallow, a tube is valuable.

I think we can tell a little sooner now when tracheotomy is indicated. When I see a patient come into the hospital, salivated and thrashing about, I want a tracheotomy whether or not he is cyanotic, because I know cyanosis will appear. Before the days of tracheotomy I never saw such a patient who did not do badly.

A cyanotic flush is bad, cyanotic pallor is worse. I think we must stress the necessity of staying away from opiates and barbiturates. I think they are deadly. The best sedative I know of is tracheotomy, oxygen, sometimes the respirator. We are not urging

tracheotomy, but the principle of keeping the airway open. It is true that in a place where only inexperienced help is available one will have to do more; in spite of the experience of our own group there are still cases with which we have trouble. We did ten tracheotomies in 26 bulbar and 12 respiratory cases this year.

DR. PAUL HOLINGER: Certainly these papers are timely expositions of important work that has been done. The paper Dr. Gray presented summed up the problem better than anything I have heard. Dr. Galloway's work, of course, is known throughout the country and the world. I think tracheotomy is being accepted more than it used to be in tracheobronchitis, and gradually its use is being expanded to other fields. Tetanus, as Dr. Galloway said, has been an indication for tracheotomy at County Hospital, and at Research Hospital we have advocated and used it with success in maintaining the airway. In frequently recurring postoperative pulmonary collapse, especially following esophageal surgery, tracheotomy is being used. In cases of cerebral hemorrhage with inability to swallow, where the patient has aspirated fluids, it should also be considered.

The increased acceptance of tracheotomy has been in part due to great improvement in postoperative care of the tracheotomized patient. The operation is no longer associated with high mortality. Steam rooms, electric suction pumps, chemotherapy, and good nursing care have all combined to almost eliminate the problem of post-tracheotomy atelectasis and pneumonia. These complications formerly led physicians to postpone tracheotomy until the patient could hardly be expected to recover, and the operation was too frequently regarded as only a last minute gesture.

I would like to correct one impression Dr. Galloway seems to have with regard to our work with carbon dioxide as an expectorant and its theoretical use in laryngotracheobronchitis. This work was done four or five years ago when we used various chemical agents to determine the effect of expectorants, oxygen and carbon dioxide on the tracheobronchial tree. We found different actions of these chemicals at various levels of the bronchial tree. We found that carbon dioxide liquefied secretions and oxygen had a drying effect. We tried this in conditions such as postoperative collapse where there was considerable secretion and also in tracheobronchitis, and confirmed the experimental studies of the various effects of these gases. This has been misinterpreted by some who thought we were advocating the use of carbon dioxide in dyspneic or even cyanotic infants who had tracheobronchitis. This is an error, and I take this opportunity to correct the statement.

DR. FREDERICK HILLER: About four or five years ago I made a few comments to your Society regarding some of Dr. Galloway's cases of tracheotomy. At that time so much could be said about the procedure, even though it appeared to have saved the life of one or another of the patients. Meanwhile you have heard Dr. Galloway discuss his impressive series of cases. I think there is no doubt that he has saved the lives of a number of patients who would have died without tracheotomy.

Neurologists are seldom asked to see such patients these days, although we are much interested in the problem. We diagnose bulbar poliomyelitis on the ground of paralytic medullary complications, and we see these patients becoming asphyctic, whereby abnormal secretions and drowning in their own lungs often complicates the picture. From a neurologic angle I think it should be possible to make a diagnosis of a primary polioencephalitis on the basis of characteristic cranial nerve paralyzes pointing to the medulla oblongata. In the older literature between 1900 and 1910 you will find such cases described. The characteristic abnormal secretion, which is not only pharyngeal but also bronchial, has also been observed. Many years ago pathologists, in describing severe inflammatory lesions in the medulla oblongata, stated that, whereas the spinal form of polio shows affinity of the inflammatory and destructive process for the ganglion cells in the anterior horn, in the bulbar type the inflammatory lesions are found to be more diffuse throughout the medulla. Lately, Dr. Magoun and Dr. McCarter have verified these observations made by Wickman and others 40 years ago.

I think there is no doubt that there are cases of bulbar poliomyelitis where respiratory difficulties leading to anoxia and hypercapnia cause a secondary circulatory damage to the brain. Whether the whole bulbar syndrome is anoxic by nature is another question. Patients who have true bulbar poliomyelitis are, in my opinion, endangered primarily by paralysis of the respiratory center to which, of course, is added the effect of respiratory paralysis itself.

I would like to propose another idea that may explain part of the clinical picture. What is the difference between acute bulbar poliomyelitis and acute bulbar paralysis as we see it, that is, in apoplectic bulbar paralysis or in cases of myasthenia gravis with severe involvement of the muscles of the pharynx and larynx? The outstanding difference seems to be this abnormal secretion in polio, which is obviously of central origin. The hypersecretion, with which the patient cannot cope, makes bulbar poliomyelitis resemble other conditions where tracheotomy is used efficiently. The hypersecretion in which patients with bulbar poliomyelitis may drown is, in my opinion,

of central origin, probably due to an inflammatory lesion about the salivatory nucleus.

We know many instances where anoxia of the brain manifests itself, e.g., in severe hemorrhages, but also under various other circumstances. From a pathologic angle, bulbar poliomyelitis cases fail to show the typical signs of anoxia and certain other manifestations which he did not describe as, for instance, loss of consciousness and convulsions, which are usually not seen in our patients. I think that hypercapnia may be more important a factor than anoxia and, if I understand Dr. Galloway correctly, his thoughts are very similar.

Would it be of any advantage to give these patients alkalies, in the form of ammonia or otherwise, to counteract the acidosis?

DR. JOHN S. GRAY (closing): Dr. Hiller asked about the possibility of giving alkalies for acidosis. Of course it would be possible to give sodium carbonate, for example, not an ammonium compound. Giving alkalies will not reduce the high level of free carbon dioxide in the blood, nor correct the anoxemia, although it might reduce the acidosis. I think to correct the latter without correcting the other would accomplish very little.

DR. THOMAS C. GALLOWAY (closing): We are firmly convinced that in the majority of cases bulbar poliomyelitis should be treated chiefly as a problem in secretional obstruction until some direct attack upon the virus is possible.

I am glad Dr. Holinger agrees that the administration of carbon dioxide is not indicated in respiratory obstruction. Tracheotomy, of course, is not a dangerous thing, as Dr. Holinger pointed out, and has many widening uses. On occasion, laryngologists stand more in need of education than do the general men as to the necessity and safety of this measure.

Dr. Hiller has brought up the question of pathologic and clinical correlation. It is a long story and the answer is not easy. From an extensive experience with monkeys and human material Dr. Bodian of Johns Hopkins says the attempt can easily lead to serious error. It must be remembered that patients with polio who come to the autopsy table have nearly all died of asphyxia. It is hard to separate virus from asphyctic effects on the central nervous system which may markedly increase secretions. We do use the bronchoscope, but in a patient in extremis, who is resistant, it may add too much strain. I have come back to feeling that it is often best to do a tracheotomy immediately rather than attempt to insert a bronchoscope. The bronchoscope does help to prevent emphysema.

**Heating of the Human Maxillary Sinus by Microwaves\***

JOHN J. BALLENGER, M.D.

AND

STAFFORD L. OSBORNE, M.D. (By Invitation)

(Abstract)

This study gives the results of experiments undertaken to heat the human paranasal sinuses.

Thermocouples were made from constantan and copper wires soldered into the tip of a Holmes antrum catheter. The couple was introduced into the anatomic ostium of the maxillary sinus without anesthesia. Temperature readings were taken frequently until a base level was reached and maintained for 20 minutes. Removing the couple the area was irradiated with microwaves for 20 minutes, after heating temperature readings were recorded. The average rise in temperature was small ( $1.35^{\circ}$  F.) but statistically significant on a 5% level.

## DISCUSSION

DR. STAFFORD L. OSBORNE: The only known factor that is really definitely proven is that high frequency current will and does produce heat in living tissue. How much is always a question. Various methods have been used to see whether or not the temperature can definitely be raised. Our work has shown some slight rise in temperature, not very marked, but still statistically significant on a 5% level of probability. One wonders whether such a slight elevation might have any clinical value. I think we forget that the heat capacity of the tissues is high; that they will absorb tremendous amounts of heat without showing a change in the basic level. It is quite conceivable to me that we might get excellent clinical results without a large increase in temperature. Increased temperature does increase blood flow. I am a little more concerned with the propaganda we shall inevitably find as a result of this new form of radiation. It should not be associated with the term "radar."

With the use of conventional diathermy radiation was measured in terms of 50 to 750 meters in wave length. This was succeeded by short wave diathermy with a wave length of 24 to 6 meters, which is now meeting competition with this new form of radiation. Most of us knew experimental work was being done with wave lengths of one meter, but believed this was merely experimental laboratory

\*This paper has been accepted for publication by the Archives of Otolaryngology.

work and would never have clinical application. With the advent of World War II, the magnetron tube was perfected giving a wave length between 12 and 13 cm. Short wave diathermy has approximately a frequency of 12 million to 100 million cycles per second. We have now jumped the development of the microtherm to 3,500,000,000 cycles per second. We have never been satisfied to assume that the only effect of diathermy would be heat. The German literature has been full of other effects than heat. It was stated that certain organisms could be definitely killed with a definite wave length while others would be stimulated to further activity. Later, evidence was shown that most of the claimed effects were the results of uncontrolled research and could not be substantiated. A paper coming from an excellent center showed that when one used microwave diathermy blood flow was materially increased but that when short wave diathermy was used the blood was not increased, or if there was any change it was backward. That work was duplicated in two or three laboratories, and these results could not be duplicated.

The matter of radiation affecting the eyes is creating a good deal of attention. Having done some work in short wave diathermy I thought it would be a good idea to duplicate this work again, and we have found no ill effects when using microwaves. Since then more extensive work has shown lenticular opacities to be present when microwave radiation is used. The thing I am afraid most people overlook is that the temperature secured in these studies is beyond the temperature that should be used in practice. I do not think there is anything new about heat producing lens opacities, but I do believe the danger is not great if one uses caution. Diathermy is not innocuous. It is dangerous in untrained hands, either short wave or the newer method. A good deal more work will have to be done before we can determine its rightful place. But one point is the small size of the directors used, which greatly limits the area to be heated. For your specialty that would be an advantage, because with short wave it is difficult to get electrodes small enough. Another important factor is the matter of dosage. No one yet has been able to tell us what constitutes an accurate dose. We have no way of determining dosage other than tissue tolerance. In your specialty, I hope you will not be misled by articles coming out that state a certain output from the generator is used. You are told to set a machine at a given setting but there is no guarantee that two patients are going to absorb the same amount of energy, although the output offered is identical, and that should be clearly kept in mind. Energy absorption determines the tissue temperature. That may explain some of the difficulty we got into in determining the value of this work. We have no assurance that all our patients re-

ceived the same amount of heat. I would suggest to anyone wishing to duplicate the work that they consider the use of thermistors instead of thermocouples for taking measurements. At the present time we have to take our couples out and, after heating, reinsert them. In this way we lose a great deal of heat and probably are not getting the highest temperature attained.

*Meeting of Monday, December 5, 1949*

THE PRESIDENT, DR. OLIVER E. VAN ALYEA, IN THE CHAIR

**Physical Laws of the Mechanism Involved in the Removal of Air from the Respiratory Tract under Normal and Abnormal Conditions\***

A. C. HILDING, M.D. (By Invitation)

DULUTH, MINN.

(Abstract)

A summary of the currently accepted theory on the mechanism of the production of postoperative atelectasis is given and certain discrepancies in it are pointed out and discussed. The author's experiments bearing on the efficiency of ciliary action in handling mucus and on the determination of the cross-sectional area of the bronchial tree at different levels are described. The laws of physics governing the absorption of air from various body spaces are reviewed and illustrated by accounts of the steps involved in the absorption of an air bubble injected into the anterior chamber of the eye at cataract surgery, the absorption of air from a completely blocked sinus, from the middle ear after closure of the eustachian tube by mechanical means, and from an obstructed lobe of a lung. Consideration of the information thus obtained points to something other than absorption operating in postoperative atelectasis to remove the air, and the author stresses the fact that ciliary action is ignored in the production of this condition.

Additional experiments carried out to determine the possible relationship between ciliary action and the development of postoperative atelectasis throw some light on the more obscure aspects of this condition. A positive or negative pressure (5-40 mm of water), reaching a maximum in from ten to twenty minutes, was produced in the trachea of a freshly killed hen by a piston of mucus

---

\*This paper will appear in full in the June 1950 issue.

moving toward the laryngeal end, motivated by ciliary power. A pressure of 150 mm of water (comparable to pressures found in atelectasis) was found when several tracheas, with a mucus piston in each, were connected in tandem; the cumulative effect was manifest in the last trachea. Ciliary mechanism, rather than absorption of air, effected the production of negative pressure in the frontal sinus of an anesthetized, newly killed or newly decapitated dog. Vacuum headache is probably a manifestation of this mechanism. In postoperative atelectasis, when all the air from the affected lobe is removed, the final one of a series of mucus pistons, pushed up the air passage by ciliary action, becomes stalled and atelectasis is complete. Absorption also plays a role depending upon such things as comparative partial pressures in the venous blood and in the alveolar air, solubility rates, diffusion rates and the presence of interposed membranes.

The conclusion is reached that absorption of air from the various body structures described occurs according to the known laws of physics. Air apparently can be completely absorbed from almost any space or tissue in the body provided sufficient time is allowed and the space containing the air can collapse. If the air-containing space cannot collapse or can do so only partially, a negative pressure develops, depending upon the gas pressures present in the absorbing fluid. In obstructed portions of the lungs, in the sinuses and probably in the middle ear, another mechanism is involved in the removal of the air, namely, ciliary action and moving masses of mucus. The negative pressure in postoperative atelectasis is probably maintained solely by ciliary action.

#### DISCUSSION

DR. PAUL HOLINGER: First I should like to congratulate Dr. Hilding upon this fundamental research work that continues the excellent studies he has already published on this and allied subjects. Anyone who has performed a bronchoscopy on a patient with postoperative massive collapse of the lung realizes that the textbook picture of a mucus plug obstructing a major bronchus should be discarded. Mucus is produced in quantity throughout the tracheo-bronchial tree and there is almost as much mucus on the so-called uninvolved side of the chest as on the side that is collapsed. No single plug is ever demonstrated as such. I wonder if Dr. Hilding's experiments can explain the rapidity of development of postoperative atelectasis in some cases—for example, those that occur on the operating table during the surgical procedure. We have x-rays that show that rapid development, and just as rapid clearing following adequate aspiration. I believe the secretions themselves can replace

a great deal of the air in the bronchi and lungs, just as Dr. Hilding said occurred in some of his experiments on the sinuses of dogs. Possibly this mechanism and the one described by Dr. Hilding work in combination to bring about the final picture.

This was an interesting presentation, and I want to thank Dr. Hilding.

DR. THOMAS C. GALLOWAY: We are very fortunate to hear this presentation by Dr. Hilding, not only for the actual scientific data he has given us, but as an example to young men who complain, with all their fine laboratory facilities, that they are not able to do good experimental work. I think it would interest them to know that Dr. Hilding does this work practically on the kitchen table with his son assisting him, procuring his own material with practically no facilities except what he improvises.

This is a difficult paper to discuss without having had a chance to sit down with a slide rule and figure out some of his computations and analyze them more completely. His ideas are scientific. There are two ways of approaching the subject; there are some interesting essays Dr. Hilding would like to read; there are formulas developed from models. There is one formula that may explain some obstruction in the divided lumina of the respiratory tract. It is not the same thing to say that the lumen of the upper trachea which has volume will have the same resistance as lumina which are much more diffuse. Roughly, the response to the flow of material can vary from solid plasma to gas as light as helium. The response is something like the fourth power of the diameter, increasing, of course, inversely as the lumen narrows. I think we must consider the secretion in the bronchioles where the plug is developed which results in atelectasis. That is important. It is quite a different thing whether those plugs are in the bronchioles or whether they are secretion. I think that, as in poliomyelitis, where the obstruction is not a mucus plug but a rather thin secretion, the picture must be quite different. I like to go back to the experiment of Steinberg in which rubber plugs were put in the bronchi and bronchioles, and it was demonstrated that atelectasis developed within 24 hours and that it became irreversible in 48 to 72 hours. It is fine to get this down to a simple formula, but when there is obstruction there are circulatory changes which add to the picture of obstruction, congestion and edema. That is something that I hope Dr. Hilding does analyze.

This is certainly a great advance in a subject about which very little that is really scientific is known. Incidentally, I think we have

all seen plugs which we have taken out with the bronchus. I remember one case of probably 3 cc of very tenacious material in the left bronchus, the removal of which relieved the atelectasis immediately. I think we have seen them in tracheobronchitis. We determine that there is no air going through, we irrigate and get out the plug and immediately we find air is going in. I feel that although we do not always see the plugs, they are likely to be important, but they are likely to be smaller conglomerations of a thinner secretion.

I think we should be grateful to Dr. Hilding for this excellent presentation.

DR. JOHN BALLENGER: I too enjoyed Dr. Hilding's paper, and I should like to make two comments. He made the statement that it takes five or six days for air injected into the anterior chamber of the eye to be absorbed, and perhaps even longer for air trapped in the sinuses or middle ear to be absorbed. Van Dishoeck's work with the pneumophone suggests that a shorter period is required for air trapped in the middle ear to disappear. In an individual with a persistently negative pneumophone reading he restored the middle ear pressure to normal by catheterization of the eustachian tube, then noted that it was only a matter of hours until the original negative pressure was regained.

The other point that occurred to me was with regard to asthmatic individuals. In the postmortem examinations of some cases, atelectasis is usually not found even though the alveoli and terminal ramifications of the bronchial tree are completely obstructed with mucus. I am familiar with the fact that some of the cilia have been destroyed by the disease process, but there are other cases in which the ciliary apparatus is intact, there is a great deal of obstructing mucus, and still atelectasis is not found. Perhaps this is explained by the anatomic phenomenon of alveolar pores. These are vents or connections between adjacent alveoli and permit exchange of air and other gases between the alveoli. It has been shown by a number of people who have worked on development of postoperative atelectasis that obstruction must occur at such a level that an entire lobe is obstructed. If the obstruction is at a lower level the air can sidetrack the obstruction by way of the alveolar pores, and atelectasis does not occur.

DR. ANDERSON C. HILDING (closing): I wish to thank the discussors for their kindly and illuminating remarks. Dr. Holinger pointed out that air could very well be displaced by the thin secretion which is often produced under anesthesia. This, I believe, is not only a possibility, but a probability.

What Dr. Galloway said about increase in friction with decreasing diameter is indeed true. It is apparently on this account that the arterial tree increases its total stream bed very rapidly as the diameter of the vessels grows smaller. If the capillaries were not 800 times the size of the aorta, the heart could not force the blood through. In the bronchial tree the situation is somewhat different. The friction factor is much less, the rate of flow of air is comparatively low and the flow is to-and-fro. For these reasons, the total cross-sectional area does not need to be as great as in the arterial tree. As he pointed out, it is a fact that the thin secretions produced in poliomyelitis are removed very slowly by ciliary action. The same is true in influenza.

#### **Fifty Years of Scientific Progress in Otolaryngology**

ALFRED LEWY, M.D.

#### **Outstanding Personalities of the First Half Century of the Chicago Laryngological and Otological Society**

SAMUEL SALINGER, M.D.

*Meeting of Monday, January 9, 1950*

THE VICE-PRESIDENT, DR. ARTHUR J. COOMBES, IN THE CHAIR

#### **Inner Ear Deafness of Sudden Onset**

JACOB J. ZUIDEMA, M.D. (By Invitation)

AND

JOHN R. LINDSAY, M.D.

(Abstract)

Sixteen cases have been presented of unilateral inner ear deafness of sudden onset with or without vestibular symptoms. The first four cases represent types in which the ear complication was associated with a systemic disease.

The next 12 cases of unilateral sudden inner ear deafness with varying degrees of vestibular disturbance are of uncertain etiology. It cannot be definitely assumed that all 12 cases have the same etiology but the similarity of the clinical picture strongly suggests that they are the same type.

Vascular disease such as hemorrhage, thrombosis and occlusion, vasomotor disturbance, allergy and toxic neuritis were all considered as being the possible etiology. Toxic neuritis must be seriously entertained as the etiologic factor, because of the high incidence in young adults below 30 years of age in the absence of chronic systemic disease.

The last three cases demonstrate a specific syndrome of bilateral inner ear dysfunction associated with an inflammatory ocular disturbance.

#### DISCUSSION

DR. FRANCIS LEDERER: If we were to take a census of this audience, I dare say everyone in practice could find among his patients 16 cases which would typify the representative ones reported. The only thing we would have to make sure of was that we were not all reporting the same cases, because patients of this type are the ones who go from one otologist to another seeking reassurance: "Will this happen to my good ear?" And you, stroking your chin and looking out of the window, say, "No, in my experience it will not happen in the other ear." You are not much of a prophet, because later they come in with tinnitus and, under your very eyes as it were, develop total deafness. You use some terms like "virus" disease, "allergy," "hemorrhage into the labyrinth," or etiologic factors for which you have not much proof. In these cases we always take x-ray films of the internal meatus. We are looking for an acoustic neuroma, but by and large these are not patients on whom you can make a specific diagnosis.

I wish we had some temporal bones to study in this series. In this disease, whatever its cause, we are dealing with an irreversible reaction, and I feel like a minister bringing a Divine wish that the same thing will not happen to the other ear. I had hoped that Dr. Lindsay and his associates would bring us something of value in the management of this condition. We are giving thiamin chloride, prostigmine, histamine, antihistamine, for the psychogenic influence on the patient, but the value has been therapeutically insignificant.

DR. THOMAS C. GALLOWAY: I wonder if the last cases cited might fall into the classification of Cogan's syndrome of interstitial

keratitis with auditory and vestibular disturbance? We recently saw a young woman with loss of hearing in both ears and recurring keratitis said to be due to this unusual disease. We are assured that the prognosis is grave.

### **Deafness Following Head Trauma: A Clinical and Experimental Study**

HAROLD F. SCHUKNECHT, M.D. (By Invitation)

(Abstract)

Diminished hearing acuity may follow a blow to the head. In some of these cases there is a fracture of the temporal bone. The fracture may be either longitudinal or transverse to the long axis of the petrous pyramid.

A longitudinal fracture of the temporal bone is characterized by bleeding from the ear, rupture of the tympanic membrane, and a combined conduction and nerve type deafness. The conduction deafness is caused by direct injury of the middle ear structures and is partially or completely reversible.

Following a transverse fracture there is severe vertigo with nystagmus to the opposite side, hematotympanum, complete deafness and total loss of vestibular function in that ear. The fracture line extends through the inner ear and degeneration of the membranous labyrinth occurs.

Deafness may also occur in an ear in which there is no clinical or roentgenologic evidence of fracture of the temporal bone. The hearing loss is of the nerve type and most severe for the high tones, particularly the 4096 frequency. In severe injury all frequencies may be involved. This type of deafness is frequently seen in the ear on the side opposite to a temporal bone fracture.

Brunner, Stenger, and Wittmaack have shown in animal experiments that hemorrhage often occurs into the perilymphatic spaces. Bleeding into the auditory nerve is less common. Wittmaack found degeneration of the hair cells and spiral ganglion to be most severe in the middle cochlear coil in cats subjected to head blows. He conjectured that a traveling pressure wave injured these structures directly.

A number of human temporal bones have been described in the literature of cases of deafness following head injury without fracture. Some have shown connective tissue and bone in the perilymphatic

spaces with degeneration of Corti's organ and the spiral ganglion. Several have shown only severe degeneration of Corti's organ and the spiral ganglion. Isolated chip fractures may also occur.

The case histories and audiograms of seven cases are presented.

Our interest was mainly in determining the nature of the pathology accounting for the nerve type deafness in such cases, so-called inner ear concussion.

The problem was studied in cats conditioned to pure tone stimuli so that hearing tests could be performed on them. These animals were subjected to head blows while under anesthesia. All degrees of deafness were produced. The 3000 to 8000 frequency range was most severely involved, paralleling that found in the human.

The histology on these animals is not complete and will be reported in detail at another time.

DR. SHERMAN SHAPIRO: There have been quite a number of experiments, beginning with those of Wittmaack, to show the histologic changes following a blow on the head in guinea pigs and other animals. These are the first I know of where conditioned reflexes have been used. I notice that the essayist mentioned what happens in the cat, and I should like to caution against translating this too literally in terms of human beings. I have had a large experience in handling head injuries, all studied from the acoustic and vestibular standpoint. My routine is to do tuning fork tests with audiograms in certain cases, and my experience is that not more than 5 or 10% show longitudinal or cross fractures, or anything involving the peripheral endings of the eighth nerve. Less than 5% complain of hearing difficulties but nearly all, in the first few months, complain of dizziness. I have found a number of times pure concussion deafness without evidence of fracture, but that is quite exceptional. The objective findings as a rule are postural nystagmus and some imbalance of the central vestibular apparatus.

I wondered about the author's case of deafness following the use of the mallet and chisel on the mastoid process. In the old days when we used the mallet and chisel very largely for mastoid operations, I never had patients complain of subsequent deafness. I never found the trauma sufficient to injure the inner ear. Two of the cases shown had symmetric curves, quite suggestive of otosclerosis, and I do not think they should be accepted as traumatic. We must remember the difference between the skull of a human being and that of a laboratory animal. Certainly few of my concussion patients without fracture led me to believe, from their complaints or from

the cochlear findings, that they would show any such pathology as indicated by a blow on the head in these animals.

DR. PAUL A. CAMPBELL: I have nothing to add either from the viewpoint of military or aviation aural trauma. One of Dr. Schuknecht's cases interested me, however, from another point of view. This was his case in which the Spondee word-response level was some 20 decibels above the pure tone threshold level. I believe this type of test discrepancy should lead one to consider possible psychogenic origin. Of course it is well known that head injuries often lead to psychogenic disorders. If discrimination tests are performed on these individuals the hearing is often found to be much poorer than one would suspect from pure tone thresholds.

DR. ALFRED LEWY: I do not know that I understood Dr. Shapiro correctly in saying that he did not always use the audiometer in head trauma cases. He said patients usually complain of vertigo, not so frequently of loss of hearing. When they do complain of loss of hearing and it is not complete, it is difficult to determine whether it is indicative of trauma, except if they show greater loss at about 4096 cycles. That has been pretty well established as quite characteristic of trauma to the internal ear. Whether or not the recruitment phenomenon is going to be a definite point in diagnosing deafness I do not know. I recently read a report on a series of cases of sudden deafness, all of which were attributed to the inner ear; four failed to show the recruitment phenomenon. Perhaps the recruitment phenomenon may apply only to certain types of inner ear deafness.

I agree with Dr. Shapiro that the mallet and gouge used in mastoid surgery did not result in permanent damage to the ear. Patients sometimes complained of headache, but no one used a one-pound hammer or a dull instrument so we can hardly compare them with the experimental data reported.

As to prognosis in vertigo following head injury, it is generally good in young people. If it has persisted more than six months, and in patients beyond middle age, it is not good.

DR. HAROLD F. SCHUKNECHT (closing): Several of the clinical cases were unusual and interesting to us. Your comments on these cases are interesting. Case 16 was the young boy who had a severe 4096 dip following a mastoidectomy. There was fairly good high tone hearing prior to surgery. A dense sclerotic mastoid cortex was removed with a mallet and gouge. We do not imply that this type of injury is common but believe it to be rare. That the deafness was the result of a shock-pulse injury seems very likely. The other

cases under discussion, mostly young individuals, all heard well before the injury and noted deafness afterward. Careful audiograms in a soundproof room are essential to the proper evaluation of hearing in all head injured persons. Case 14 was a 54-year-old woman with a rather flat hearing loss of between 30 and 50 decibels in both ears following a head injury. A striking discrepancy was the relatively good score on the Spondee word test. We agree with Dr. Campbell that the possibility of psychogenic deafness exists in this one case. Repeated audiograms were remarkably consistent in this case. That any of these patients might represent cases of otosclerosis, Ménière's disease, etc., seems beyond the realm of possibility.

### Some Unusual Mixed Tumors of the Nose and Throat

HENRY B. PERLMAN, M.D.

The so-called "mixed tumors" of the salivary and mucous glands are of special interest to the otolaryngologist. Although the term is widely used it often represents an obscure clinical and pathologic entity, especially when applied to tumors found away from the major salivary glands. Because of the relative high incidence in the parotid gland, this diagnosis is readily considered when dealing with a tumor here, and the clinical and pathologic picture is more generally agreed upon. They are much less frequent in the sublingual gland, occurring about one in every 100 cases involving the parotid gland. These tumors do occur in the mouth, tongue, soft palate, nose, sinuses, pharynx, etc. In these less common locations a diagnosis may be suspected when confronted with a slowly expanding, rubbery, lobulated, nonpainful, nonmetastasizing encapsulated tumor. New and Withridge collected over 60 cases in the soft palate and pharynx alone, while Owens found reports of several hundred during the last 20 years, with the palate and nose being the most common site. There is a problem of preliminary biopsy too. One may hesitate to open the capsule for fear of spreading tumor cells. Furthermore, a small specimen may not reveal the true nature of the lesion, which may have benign and malignant characteristics histologically in different parts. These tumors may show bone destruction on x-ray that resembles that seen in infiltrative carcinomas, but may be found at operation to be due to slow resorption of bone on account of pressure, as well as by definite infiltration. The tumor may remain unchanged in basic character while growing to enormous size. This is commonly seen in parotid tumors. The facial nerve may be slowly stretched over such a tumor, as over an acoustic neuroma, without disrupting its function. Since they are as a rule

radioresistant, the correct diagnosis guards against unnecessary radiation, too radical surgery, or a very grave prognosis. The surgical problems concerning the identification and preservation of the facial nerve when dealing with the parotid tumor were well described recently by Buxton, Maxwell and Cooper. The use of a nerve stimulator to help identify some of the facial nerve branches at operation is also worth emphasizing.

The surgical attack is less radical than that needed for an infiltrative nonencapsulated tumor, but the capsule may be very thin in some places or, indeed, missing. The capsule should be very carefully handled to prevent opening into the tumor which grows from its inner surface. The capsule must be cleanly removed with the tumor to affect a cure.

Clinically, this is a peculiar tumor in that it may show long periods of quiescence, remaining a given size for many years and then growing more rapidly. The longest period in our group was about 30 years. Similarly, after removal years may elapse—10-20 years is not unusual in our records—before recurrence is noted. The recurrence is not necessarily of a more malignant nature either clinically or pathologically and may be localized without regional gland involvement. Several recurrences may take place in this manner. On the other hand, recurrences may take place in ever more frequent sequence after many years of apparent cure, and leading finally to local and to distant metastases with fatal termination.

This is a peculiar tumor histologically, the character of which has long been disputed, but which is now considered by most competent pathologists as arising only from epithelial cells in the gland. A single case may show many different kinds of architecture, but on the other hand may be quite homogeneous in its cell structure. The term "mixed cell tumor" or "mixed tumor" is really inappropriate and stems from the belief that the cartilage-like stroma in some of these cases represented a connective tissue participation as well as an epithelial cell origin. These cartilage-like areas in the stroma are the result of mucus formation probably by the tumor cell. The tumor cells may be widely separated in some areas of this mucoid stroma and give the appearance of immature cartilage. Another term that has been used to describe the histologic picture of some of these glandular tumors is "cylindroma" a term that is also of little significance for a basic understanding of these cases, and used by different authors with different implications. The term may be used to describe the solid cords of cells seen in some of these tumors that resemble cylinders, while another meaning pertained to

the presence of a thick collar of pink staining collagen around the columns of tumor cells.

Four cases are described in detail, one arising in the soft palate, one in the sublingual gland and two in the antrum and floor of the nose, probably arising from the inferior turbinate.

Certain modifications of the lateral rhinotomy operation used in the last two cases are described.

#### DISCUSSION

DR. STANTON A. FRIEDBERG: I think we are deeply indebted to Dr. Perlman for this presentation. It is a live and important subject to otolaryngologists because we are, by and large, the first to see these patients. This is the type of tumor that evokes more difference of opinion among pathologists than almost any other lesion in the human body.

As Dr. Perlman mentioned, there is apt to be great variation in the histology in different parts of the same tumor. The combination of epithelial and connective tissue elements has led to their being designated as mixed tumors, but we often see references to them as adenoid-cystic carcinoma, cylindroma, basal cell carcinoma, myxochondrocarcinoma, muco-epidermoid tumor of salivary gland origin, etc. Although some of these lesions may be encapsulated, this is not universally true and capsular boundaries are apt to disappear as the growths increase in size. Whereas some may be judged histologically benign, the clinical behavior certainly demands that we consider them as potentially malignant, in view of their marked tendency toward local recurrence and their reactivation after periods of quiescence. For this reason it seems to me that the classical intranasal and sublabial antrotomies are completely out of order in the surgical approach to these lesions when they involve the maxillary sinus.

A lantern slide is presented showing the operative defect in a girl who had had multiple limited operations for a mixed tumor of the palate over a period of 11 years. One year ago a resection of the maxilla was performed and, whereas we are dubious as to the eventual outcome, we feel that for the first time a truly adequate removal was effected. A prosthetic device allows her to eat and speak normally but permits of ready access to the cavity at all times. In my opinion the initial attack on these tumors will determine the outcome and I should like to make a plea for their radical removal at the first operation.

DR. MAURICE F. SNITMAN: The term "mixed tumor" is inappropriate, inasmuch as it does not give a true histologic picture of the lesion and does not sufficiently stress malignancy.

Histologically, the tumor is an epithelial structure, the presence of cartilage-like or myxomatous tissues within it being the result of a mucoid transformation of the stroma by the secretion of the acini of the tumor. The inclusion of this tumor in the adenocarcinoma group could be considered proper, in view of its well known malignant character.

When the tumor is located in the nasal cavity or antro-ethmoid area, we use the sublabial approach rather than the external facial incisions. It is imperative that a good portion of the hard palate of that side be removed so that a patent opening is obtained through which the cavity may be carefully observed postoperatively.

These tumors are malignant and will recur if incompletely removed.

DR. THOMAS C. GALLOWAY: We are likely to try to include in one category too many types of tumor. They cannot always be classified too closely. The tumors which arise in the sinuses, called by Ringertz salivary cell tumors, behave differently from those more often seen in salivary glands or soft tissues. The tumor is rather uniform grossly and histologically, is likely to be encapsulated, and destroys bone by erosion rather than invasion, and does not greatly change its histologic structure, except as modified by pressure, infection, etc.

The interesting thing about the mixed type tumor is that when malignancy arises it appears to come from multiple foci. It may remain relatively latent for years and then rather rapidly begin to grow from these numerous centers. Has the speaker any explanation?

I hope no one considers the advice to do radical surgery a direction to do slashing surgery on gland tumors. The dissection must be most meticulous. The capsule is very thin and if it is torn recurrence is likely with subsequent failure; yet for removal, dissection must be carried to this capsule.

Ten years ago I removed a so-called myxoma from the parotid of a 20-year-old girl with no recurrence to date. Her identical twin died a few years earlier after operation on a parotid tumor. Could these tumors have arisen from one anlage?

DR. SAMUEL SALINGER: I should like to add one or two points. In my experience the tumor in which the capsule is ill-defined, and

in which the epithelial cellular elements predominate, is the one in which you are likely to get malignant degeneration or recurrence. The five-year limit is not always the outside limit. Quite recently one of our patients had a recurrence in the same location after eight years. As a rule tumors in the upper jaw usually recur locally, whereas tumors of the mandible are more likely to metastasize.

The point Dr. Galloway brought out I think is very appropriate. There are a variety of tumors bearing various names according to the predominating elements, and yet all genetically the same. The important thing is not the name but the recognition of the potentiality of the growth.

DR. HENRY B. PERLMAN (closing): The question of the amount of resection one must practice in these tumors is difficult to settle, but I think you would approach a tumor like this less radically than one which is wild from the onset and, while we have to be radical to the extent that the exposure technique in the last two cases was radical, the actual tissue removed was limited to the tumor itself, rather than removing a margin of normal looking tissue as in infiltrating carcinomas.

The terminology of these tumors is very confusing, and I would not attempt to commit myself on the histologic sections. We depend on the pathologist, who has cooperated with us for many years; we feel her judgment has great merit and have come to depend upon it, because she knows the clinical course of these cases almost as well as we do. We depend upon her to point out histologic features which may be clinically significant.

Leaving an opening into the mouth might be the soundest policy, certainly in attacking one of the infiltrating nonencapsulated antral carcinomas where you can get recurrence from multiple areas. The reason for preserving the integrity of the mouth cavity in this third case was that it appeared to be a circumscribed encapsulated tumor. So far as the histogenesis is concerned, the more you read about these tumors the more you realize that it is a specialized field of histopathologic interpretation; a clinician may rightfully hesitate to make a statement about the histologic sections. A clinician may have ideas about the histology but a trained pathologist with a large experience with the clinical and pathologic nature of tumors in this field can render valuable aid. After obtaining consultation with the pathologist and the radiologist, the clinician must make the ultimate decision as to the best method of procedure in any given tumor.

We do not know why these tumors may remain latent for 10 or 20 years and then start growing. The recurrence may not be more malignant in character than the original tumor.

Neural infiltration is one of the histologic features that suggests a method of spread of tumor that is dangerous to the patient. Obviously, five-year cures do not mean anything in mixed tumors.

## Abstracts of Current Articles

---

### EAR

#### Developments in the Surgery of the Labyrinth.

*Brownlie-Smith, A.: Edinburgh M. J. 56:256-264 (June) 1949.*

The author first discusses the development of surgery of the infected labyrinth describing the operations of Jansen (1893), Hinsberg (1902), Botey (1903), Bourguet (1905), and Richards (1907).

The remainder of his paper has to do with surgery of the non-infected labyrinth which originally came into being in the effort to improve the hearing of patients suffering from otosclerosis. To this end Kessel of Jena in 1876 removed the footplate of the stapes. In 1896 Passow attempted to replace the fixed stapes by a movable membrane and to this end drilled into the promontory with a trephine. He improved the hearing but the risks of infection were great. In 1906 Barany suggested an opening into the posterior canal in order to prevent infection. The success he achieved was only temporary. In 1913 Jenkins of London fenestrated two patients. In one he covered the fistula with a skin graft and in the other with a small flap from the membranous meatus. He abandoned the method because one of the patients became totally deaf and in the other the hearing became worse.

Holmgren of Sweden fenestrated his first patient in 1917 using the superior canal in the hope that the middle fossa dura would prevent the regrowth of bone. Sourdille of Nantes operated in three or more stages and produced some lasting results. In 1917 J. S. Fraser of Edinburgh did a radical mastoid operation removing the bony cap from the lateral canal and covering it with a skin graft. The patient obtained improved hearing in the operated ear. In the second case the hearing was completely lost because of labyrinthitis. Some years later he operated on a third noninfected labyrinth but the patient developed meningitis and died. In 1937 Brownlie-Smith himself operated on a patient in two stages. The original hearing improvement was later lost and a revision was done with improvement in hearing again. Seven years later the hearing had once again deteriorated and a second revision was done with improvement of the hearing.

Lempert of New York has introduced the transmeatal approach to the lateral canal. Hall of Scotland uses a drill, continuous irrigation and suction to remove the bone dust and makes the fenestra with the aid of a dissecting microscope. The author at this point discusses the advent of the binocular dissecting microscope.

Operative work on the labyrinth today is mainly confined to the treatment of two diseases: one, the deafness resulting from otosclerosis and two, Ménière's disease. Regarding the treatment of otosclerosis the author discusses the pioneer work of Holmgren, and his own series of 64 cases with one death and immediate improvement of hearing in 46 cases. He discusses the efforts of Holmgren to prevent the bony closures of the fenestra by the use of radium, gold leaf, and periosteum, the efforts of Lempert in changing the position of the fenestra from the prominence of the lateral canal to the surgical dome of the vestibule, burnishing the edges of the fenestra with lead and the use of a cartilage stoppel. He calls attention to Hall's technique of trimming down the bone of the labyrinth capsule and of removing the bone of the actual fenestra in one piece to prevent the development of bone dust and fine slivers of bone. In spite of all these efforts the author states that bony closure of the fenestra, while not as frequent as formerly, still occurs in a fairly high percentage of cases.

In order to keep the fenestra from closing, Brownlie-Smith suggests inserting into the fenestra a small mastoid cell which has been prepared from the patient's own mastoid and which has a preformed opening, into the prepared fenestra in the hope that the bone of the mastoid canal would fuse with the edges of the fenestra. He has used such a bone graft in one case. He voices some question whether such a bone graft would live or whether it would die and become a sequestrum.

*Operative Treatment of Ménière's Disease.* In this connection the author mentions the injection of absolute alcohol into the labyrinth through the tympanic membrane and round window or through an opening made into the lateral canal as in a fenestration operation. The labyrinth can also be destroyed by removing the membranous labyrinth through a slightly larger fenestra by the method of Cawthorne. He believes that there is more danger involved for the facial nerve from the alcohol injection than from the operation of Cawthorne.

"Nerve" Deafness: Its Clinical Criteria, Old and New.

Dix, M. R., Hallpike, C. S., and Hood, J. D.: *Proc. Roy. Soc. Med.* 42:527-536 and 540 (Sect. Otol. pp. 11-20 and 24) (July) 1949.

The tuning fork tests of Schwabach, Rinne and Weber are sufficient to effect a reasonably clear-cut distinction between perceptive and conductive deafness but these "old" criteria of nerve deafness have one limitation in that while they will diagnose the existence of nerve deafness they do not indicate in which way particular components of the perceptive mechanisms have been affected.

The "new" criteria which have been developed within the last ten years, namely, the Loudness Recruitment Test and the Intelligibility Test for Amplified Speech, add to the diagnosis of nerve deafness some insight into the nature and localization of the pathological process within the perceptive mechanism. These newer tests have been particularly applied to two groups of cases; first, the Ménière's disease of the labyrinth and second, eighth nerve neurofibroma; for we know that in the first, the pathological disturbance is in the cochlea while in the second, it is a degenerative process in the eighth nerve fibers.

The results of the classical tuning fork tests, the old criteria, reveal little to distinguish these two groups of pathological conditions. The classical tuning fork tests in eighth nerve neurofibroma as in Ménière's disease yield only the conventional picture of perceptive deafness.

There is general agreement by all who have investigated the loudness recruitment phenomenon that it is absent in uncomplicated conduction deafness and it is present in a wide variety of disorders of the internal ear and eighth nerve. So the test is generally supposed to simply add weight to the findings of the tuning forks.

"In Ménière's disease with disorder of the hair cells, loudness recruitment is always present. It is generally complete and over-recruitment may be present. In eighth nerve neurofibroma, however, loudness recruitment, hitherto described as being uniquely distinctive of nerve deafness, is characteristically not present at all. Instead, there occurs a type of response which is much more in accordance with that found in middle-ear deafness." The loudness recruitment test, therefore, makes possible a clear distinction between "nerve" deafness due to pathology in the end organ and that due to pathology of the nerve itself.

In regard to the intelligibility of amplified speech test, it was found by the authors that in both conductive lesions and in perceptive lesions of the eighth nerve neurofibroma type, intelligibility

continues to improve with amplification up to high levels but this is not true of the perceptive deafness of the Ménière or end organ type, for here "while recruitment of loudness is the rule, recruitment of intelligibility which might be expected may be conspicuously absent. In fact the opposite may appear to occur, namely, that as loudness increases intelligibility falls off."

By means of these newer concepts or criteria, it is possible now to differentiate a nerve deafness of the nerve type from one of the end organ type.

GROVE.

## NOSE

### Management of Allergic Sinusitis.

Glover, C. H.: *South. M. J.* 42:613-619 (July) 1949.

Nasal allergy, particularly the perennial type, with persistent or recurrent nasal blockage is known to be a great factor in the etiology of sinus disease.

It is no difficult matter to differentiate primary infections and allergic rhinitis if cytology of the nasal smears is given its deserved consideration. This alone will throw light on the etiology of symptoms and Hansel says that this test should be made on all patients with nasal symptoms.

Instances of nasal disease associated with pathologic changes in the nasopharynx deserve serious consideration. The nasopharynx will oftentimes be found to contain a large amount of hyperplastic tissue resembling adenoid growth and is often removed by the surgeon as such, only to have a return of this tissue in a short length of time. This tissue is prone to become infected and the erroneous diagnosis of chronic streptococcus throat made and treatment with a sulfa drug or penicillin instituted, only to have a recurrence of the condition which finally invades the paranasal sinuses, larynx and bronchi.

Allergic therapy should be instituted as soon as allergic substances are demonstrated. Desensitization with antigens according to titration method advocated by Rinkel, and vaccines which show varying degrees of sensitivity along with dietary restriction, is the treatment of choice.

Food, as an etiologic factor in perennial nasal allergy, is of great importance due to the predisposition to sinus disease. Allergic

foods must be excluded from the diet if good clinical results are expected. Inhalants, like foods, deserve the same consideration.

TAYLOR.

## LARYNX

### New Operative Procedure for Laryngofissure.

*Broyles, Edwin N.: South. M. J. 42:825-827 (Oct.) 1949.*

Intubation with a tracheal tube is done after sodium pentothal anesthesia or, if desired, a tracheotomy is done first under anesthesia. The skin of the neck is cleaned with soap and water, followed by alcohol and ether. A midline neck incision is made from the thyroid notch to the sternal notch. The isthmus of the thyroid gland is clamped and divided, exposing the tracheal rings. The external perichondrium of the thyroid cartilage is incised in the midline and gently elevated. On the diseased side, this is carried far posterior, while on the unaffected side, it is carried back only 1.5-2 cm. When this is completed, a mastoid curette is used to peel away the cartilage exposing the membrane of the intralaryngeal muscles. This resection of the cartilage is then extended past the midline, above and below the anterior commissure attachment, to the opposite side, leaving a small piece of cartilage to which the anterior commissure tendon is attached. If the thyroid cartilage has ossified, bone rongeurs may have to be used. The window is carried far back on the diseased side. Interrupted No. 000 chromic catgut sutures outline the resection on the good side, and an incision is made inside the suture line. The resected flap is turned back and the interior of the larynx and the growth inspected. If it is of such size that it can be completely removed, along with a margin of normal tissue, the sutures are placed as the incision is carried back on the affected side. Routinely this should extend to, and include, the vocal process of the arytenoid, the false cords, ventricle and true cord. The complete resection, then, is the diseased cord, ventricle and false cord, the anterior commissure and the anterior third of the good cord.

The perichondrium is closed with interrupted sutures. The extralaryngeal muscles, subcutaneous tissues and the skin are likewise closed.

A tracheotomy tube is left in place for 24-48 hours.

This technique does away with the troublesome handling of cartilage and changes it to a soft tissue operation.

TAYLOR.

## BRONCHI

## Solitary Cerebral Metastases from Bronchial Carcinomata. Their Incidence and a Case of Successful Removal.

Flavell, Geoffrey: *Brit. M. J.* 2:736-737 (Oct. 1) 1949.

The only successful removal on record, except for the case the author reports below, of both primary and secondary cerebral growths was done by Thurel in 1942. The incidence of cerebral metastasis in carcinoma of the lung was estimated at 16.5% by Ochsner and DeBaKey, reviewing 3047 patients. Other authorities put the figures even higher—Graham and Wagner at 24% and Olsen at 36%.

Flavell then reports the case of a male, aged 34, who on routine radiography was found to have a circumscribed shadow at the hilum of the left lung in June, 1947. In October, 1947, he developed headache followed by an epileptiform seizure and incontinence of urine and later diplopia. On hospital admission he exhibited slight papilloedema, slight left lower facial weakness but no other signs either in his chest or nervous system. Bronchoscopy was negative but in spite of this it was felt that there was a primary carcinoma of the lung accompanied by a cerebral metastasis. Ventriculography showed a displacement of the system to the left and no filling of the right ventricle.

On January 8, 1948, a large lobulated vascular mass was removed from the right temporal lobe. Histological sections proved it to be a secondary carcinoma. Eight weeks later a left pneumonectomy was carried out. A massive growth was found in the hilar region but no enlarged mediastinal glands were found. The tumor was a squamous cell carcinoma.

At the time of publication, 18 months after the pneumonectomy, the patient was well and had no sign of any recurrence or any further metastasis.

GROVE.

## MISCELLANEOUS

## Serological Classification of Viridans Streptococci; from Subacute Bacterial Endocarditis, Teeth and Throats.

Selbie, F. R., Simon, Rosemary D., and Robinson, R. H. M.: *Brit. M. J.* 2:667-672 (Sept. 24) 1949.

Streptococci of the viridans type from cases of subacute bacterial endocarditis and from normal throats and extracted teeth were differentiated into five serological groups.

The distribution of the five serological groups of the streptococci from acute bacterial endocarditis is similar to that of the streptococci from throats and teeth, indicating that the streptococci of subacute bacterial endocarditis are derived from the region of the mouth.

Streptococci isolated during relapses after penicillin treatment in subacute bacterial endocarditis are usually similar in their serological and biochemical characters to the streptococci isolated before treatment, indicating that relapses are gradually due to recrudescence of the original and not to re-infection by another streptococcus.

GROVE.

**Aureomycin Treatment of Herpes Zoster.**

Finland, M., Finnerty, E. F., Jr., Collins, H. S., Baird, F. W., Gocke, T. M., and Kass, E. H.: *New England J. Med.* 241:1037-1046 (Dec. 29) 1949.

Finland and his co-workers present the results of the treatment with aureomycin in 24 cases of herpes zoster. Inasmuch as six of these had involvement about the face or ears, the findings should be of interest to otolaryngologists. Aureomycin appeared to halt the progress of the disease and to bring about rapid healing of the lesions. Pain accompanying the acute lesion subsided after the first day of treatment. Post-herpetic pain was not prevented in patients in whom treatment was begun more than two weeks after onset of the disease. The oral dosage was 4 gm daily, divided into four doses, until lesions were well dried. Then half this amount was given for three to five additional days. Aureomycin was injected intravenously if oral administration could not be tolerated.

HILL.

**Generalized Intestinal Polyposis and Melanin Spots of the Oral Mucosa, Lips and Digits.**

Jeghers, H., McKusick, V. A., and Katz, K. H.: *New England J. Med.* 241:993-1005 and 1031-1042 (Dec. 22 and 29) 1949.

This is an exhaustive review of ten cases of a syndrome of melanin spots in the buccal mucosa, lips and digits associated with polyposis of the small intestine. The authors feel that this syndrome is a distinct clinical entity. Evidence is presented demonstrating its hereditary nature. Round, oval or irregular patches of brown or almost black pigment are seen on the lips and buccal mucosa, occasionally on the gums or hard palate and rarely on the tongue. Inasmuch as the outstanding feature of the cases is the oral pigmentation, it would seem advisable for otolaryngologists to be conversant with the syndrome.

HILL.

## Books Received

---

### **Chirurgie de l'Oreille, du Nez, du Pharynx et du Larynx.**

By *Maurice Aubry, Laryngologiste des Hôpitaux de Paris.* 4th Ed. Pp. 965 with 729 illustrations. Paris, Masson & Cie., 1949. (Price 3000 fr.)

### **The Temporal Bone and the Ear.**

By *Theodore H. Bast, A.B., Ph.D., Professor of Anatomy, University of Wisconsin, Madison, Wisconsin, and Barry J. Anson, M.A., Ph.D., Professor of Anatomy, Northwestern University Medical School, Chicago, Illinois.* Pp. xviii+478, with 165 illustrations. Springfield, Charles C. Thomas, 1949. (Price \$12.00)

### **Fundamentals of Otolaryngology. A Textbook of Ear, Nose and Throat Diseases.**

By *Lawrence R. Boies, M.D., Clinical Professor of Otolaryngology, Director of Division of Otolaryngology, University of Minnesota Medical School, and Associates.* Pp. xv+443, with 184 figures. Philadelphia and London, W. B. Saunders Company, 1949. (Price \$6.50)

### **Atlas of Oral and Facial Lesions and Color Film Library.**

By *Ralph Howard Brodsky, D.M.D., Consulting Oral Surgeon, Department of Hospitals, New York City; Lecturer in Stomatology, Graduate School of Medicine, New York University; Associate Dentist to the Mt. Sinai Hospital, N. Y.; Founder and Executive-Secretary, Pan American Odontological Association.* Pp. x+127 with 100 figures and corresponding color slides. Baltimore, The Williams and Wilkins Company, 1948.

### **Pollen Slide Studies.**

By *Grafton Tyler Brown, M.D., F.A.C.P., Instructor in Clinical Medicine, Georgetown University School of Medicine; Consultant on Allergy, United States Public Health Service; Head, Division on Allergy, Doctors Hospital; Washington, D. C. With a forward by Wallace M. Yater, M.S. (In Medicine), M.D., F.A.C.P., Director, Yater Clinic; Civilian Consultant, Army Institute of Pathology and Walter Reed General Hospital; Formerly, Professor of Medicine, Georgetown University School of Medicine; Washington, D. C.* Pp. xii+122 with 98 figures. Springfield, Charles C. Thomas, 1949. (Price \$6.00)

### **Audiology. The Science of Hearing. A Developing Professional Specialty.**

By *Norton Canfield, M.D., Associate Professor of Otolaryngology, Yale University School of Medicine; Board of Directors, Audiology Foundation; Board of Directors, American Hearing Society; Vice-President, International Audiology Conference; Consultant in Audiology, Veterans Administration; Consultant in Otolaryngology, Office of the Surgeon General, Department of the Army, Washington, D. C.* Pp. ix+45. Springfield, Ill., Charles C. Thomas, 1949. (Price \$1.75)

**Le Traitement des Tumeurs Malignes Primitives du Maxillaire Supérieur.**

By M. Dargent, *Chirurgien du Centre Anticancéreux*; M. Gignoux, *Oto-rhino-laryngologiste des Hôpitaux*; and J. Gaillard, *Chef de Clinique O.R.L. à la Faculté, Lyon*. Pp. 216, with 39 figures. Paris, Masson & Cie., 1949. (Price 500 fr.)

**Radiologic Exploration of the Bronchus.**

By S. Di Rienzo, M.D., *Assistant Professor of Radiology and Physiotherapy; Chief of the Radiology Department of the Institute of Cancer, The University of Cordoba, Argentina*. Pp. xiv+332, with 466 figures. Springfield, Charles C. Thomas, 1949. (Price \$10.75)

**Ricerche morfologiche sull'apparato di trasmissione del suono. Sound-conducting apparatus: a study of morphology.**

By Zaccaria Fumagalli, translation by O. Speciani. *Archivio Italiano di Otolgia, Rinologia e Laringologia*, Vol. LX, Suppl. 1, 1949, 323 pp.

**Introduction a l'etude de la chirurgie de la surdite ou cophochirurgie.**

By G. L. Hicquet and M. Van Eyck. *Acta Oto-rhino-laryngologica Belgica* 1:421-629, 1947.

**Blakiston's New Gould Medical Dictionary.**

Editors: Harold Wellington Jones, M.D., Normand L. Hoerr, M.D., and Arthur Osol, Ph.D. Pp. xxviii+1294, with 252 illustrations on 45 plates, 129 in color. Ed. 1, Philadelphia-Toronto, The Blakiston Company, 1949. (Price \$8.50)

**Clinical Audiology.**

By Maurice Saltzman, M.D., *Assistant Professor of Otorhinology, Temple University School of Medicine; Associate Otolaryngologist, Mount Sinai Hospital, Philadelphia; Diplomate, American Board of Otolaryngology*. Pp. xiii+195, with 82 figures. New York, Grune & Stratton, Inc., 1949. (Price \$5.00)

**Recent Advances in Oto-laryngology.**

By R. Scott Stevenson, M.D., Ch.B., F.R.C.S., *Surgeon, Metropolitan Ear, Nose and Throat Hospital; President, Section of Otolgy, Royal Society of Medicine*. 2nd Ed. Pp. viii+395, with 8 plates and 106 figures. Philadelphia, The Blakiston Company, 1949. (Price \$6.00)

**A History of Oto-laryngology.**

By R. Scott Stevenson, M.D., F.R.C.S. (Ed.) and Douglas Guthrie, M.D., F.R.C.S. (Ed.) Pp. vii+155, with 52 figures. Baltimore, The Williams and Wilkins Company, 1949. (Price \$5.00)

**Hearing Tests and Hearing Instruments.**

By Leland A. Watson and Thomas Tolan, M.D. Pp. x+597, with 239 figures. Baltimore, The Williams & Wilkins Company, 1949. (Price \$7.00)

**Theory of Hearing.**

By Ernest Glen Wever, Ph.D., *Professor of Psychology, Princeton University*. Pp. xiii+484, with 137 figures and 8 tables. New York, John Wiley & Sons, Inc., 1949. (Price \$6.00)

## Notices

---

### AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations in 1950:

May 17-20 in San Francisco, California, at the Hotel Mark Hopkins.

October 3-6 in Chicago, Illinois, at the Palmer House.

---

### PACIFIC COAST SOCIETY

The Pacific Coast Society will hold its annual meeting at the St. Francis Hotel, May 27-31, 1950. Members of the National Societies convening in San Francisco are cordially invited to attend and participate in the discussions.

---

### ANNOUNCEMENT

The University of Minnesota announces the 7th biennial Continuation Course in Otolaryngology to be held June 26, 27, 28, 29, 30, 1950. This Course is designed to bring to the practicing otolaryngologist the newer concepts and developments in the specialty. The Course will be under the direction of Dr. Lawrence R. Boies and associates of the University Medical School. Dr. Fred A. Figi, Dr. Henry L. Williams and others of the Graduate School Faculty will participate in the instruction. Guest lecturers will include Dr. Percy Ireland, Toronto; Dr. LeRoy Schall, Boston; Dr. Philip Meltzer, Boston; and Dr. John Shea, Memphis.

The fee for this Course is \$50.00. The enrollment is limited. Application should be made at an early date to the Director, Center for Continuation Study, University of Minnesota, Minneapolis 14, Minnesota.

## AMERICAN HEARING SOCIETY

## KENFIELD MEMORIAL SCHOLARSHIP

In 1937 a sum of money was subscribed in memory of Miss Coralie N. Kenfield of San Francisco, California, a teacher well known throughout the United States for her high ideals and advanced methods in teaching lip reading. This money, placed in the Kenfield Memorial Fund, is administered by the American Hearing Society and provides for an annual scholarship. The amount of the Kenfield Memorial Scholarship for 1950 is one hundred dollars (\$100.00).

Applications for the scholarship will be considered from any resident of the United States who desires to teach lip reading (speech reading) with or without other types of hearing and speech therapy to the hard of hearing, and who can meet the following requirements:

A. *Personal*

Well adjusted individual with a pleasing personality, legible lips, a good speech pattern and no unpleasant mannerisms.

B. *Education*

College graduate with a major in education, psychology, and/or speech, plus professional training in lip reading: 30 clock hours of private instruction under an approved teacher of lip reading or 60 clock hours of instruction in public school classes under an approved teacher of lip reading.

The winner of the scholarship may take the Teacher Training Course from any normal training teacher or school or university in the United States offering a course acceptable to the Teachers' Committee of the American Hearing Society. The scholarship must be used within one year from the date the award is made.

Applicants must be prospective teachers of lip reading to the hard of hearing. Those already teaching lip reading cannot be considered.

Applications must be filed between March 1 and May 1, 1950 with:

Miss Rose V. Feilbach,  
Chairman, Teachers' Committee,  
1157 No. Columbus Street,  
Arlington, Virginia.

## NOTICE

The annual congress of the German Society of Ear, Nose and Throat Specialists will be held in Bad Kissingen May 24-27, 1950. The chief topic of discussion will be "Errors and Dangers in the Management of Ear, Nose and Throat Patients." American colleagues are cordially invited.

Inquiries may be addressed to Professor H. Frenzel, Geiststrasse 10, Göttingen, Germany.

**HEARING AIDS ACCEPTED BY THE  
COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION  
THE AMERICAN MEDICAL ASSOCIATION**

(List Corrected to March 1, 1950)

<b>Aurex Model F</b>	<b>Radioear Permo-Magnetic</b>
<b>Aurex Model H</b>	<b>Multipower</b>
<b>Beltone Mono-Pac</b>	<b>Radioear Permo-Magnetic</b>
<b>Beltone Harmony Mono-Pac</b>	<b>Uniphone</b>
<b>Beltone Symphonette</b>	<b>Silver Micronic (Crystal Receiver)</b>
<b>Dysonic Model No. 1</b>	<b>Model 101</b>
<b>Electroear Model C</b>	<b>Silver Micronic (Magnetic and</b>
<b>Gem Hearing Aid Model V-35</b>	<b>Crystal) Models 202M &amp; 202C</b>
<b>Maico Type K</b>	<b>(See Micronic)</b>
<b>Maico Atomeer</b>	<b>Silvertone Model 103BM</b>
<b>Mears Aurophone Model 200</b>	<b>Sonotone Model 600</b>
<b>1947-Mears Aurophone Model 98</b>	<b>Sonotone Model 700</b>
<b>Micronic Model 101 (Magnetic</b>	<b>Sonotone Model 900</b>
<b>Receiver)</b>	<b>Sonotone Models 910 &amp; 920</b>
<b>Micronic Model 303</b>	<b>Super-Fonic Hearing Aid</b>
<b>(See Silver Micronic)</b>	<b>Televox Model E</b>
<b>Microtone T-3 Audiomatic</b>	<b>Telex Model 22</b>
<b>Microtone T-4 Audiomatic</b>	<b>Telex Model 97</b>
<b>Microtone T-5 Audiomatic</b>	<b>Telex Model 99</b>
<b>National Cub Model (C)</b>	<b>Telex Model 1700</b>
<b>National Standard Model (T)</b>	<b>Tonemaster Model Royal</b>
<b>National Star Model (S)</b>	<b>Trimm Vacuum Tube Model 300</b>
<b>Otarion, Model E-1</b>	<b>Unex Model A</b>
<b>Otarion, Model E-1S</b>	<b>Unex Midget Model 95</b>
<b>Otarion, Model E-2</b>	<b>Unex Midget Model 110</b>
<b>Otarion, Model E-4</b>	<b>Vacolite Model J</b>
<b>Paravox Models VH and VL</b>	<b>Western Electric Model 63</b>
<b>Paravox Model XT</b>	<b>Western Electric Model 64</b>
<b>Paravox Model XTS</b>	<b>Western Electric Models 65 &amp; 66</b>
<b>Paravox Model Y (YM, YC and</b>	<b>Zenith Model 75</b>
<b>YC-7)</b>	<b>Zenith Miniature 75</b>

All of the accepted hearing devices employ vacuum tubes.

Accepted hearing aids more than five years old have been omitted  
from this list for brevity.

**TABLE HEARING AIDS**

<b>Aurex (Semi-Portable)</b>	<b>Sonotone Professional Table Set</b>
<b>Precision Table Hearing Aid</b>	<b>Model 50</b>

